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# REFLEX PARALYSIS

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# REFLEX PARALYSIS:

## ITS PATHOLOGICAL ANATOMY,

AND RELATION TO THE

## SYMPATHETIC NERVOUS SYSTEM.

BY

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This Essay is mainly a reprint of a paper *On the Pathology of Reflex Paralysis, and its Relations to the Sympathetic System*, which has appeared in the *New York Medical Journal* for April and May of the present year. The additions are but mere outlines of subjects which I have discussed, since 1861, in lectures delivered at the University Medical College of New York, and in contributions to the *American Medical Times* (1861–62–63).

To Baron Egloffstein I am gratefully indebted for his kindness and valuable assistance in reproducing, in the most minute and delicate manner, by his admirable process of heliography on steel, the photographic copy and microscopic drawings of the specimens described in these pages.

New York, May, 1866.

# REFLEX PARALYSIS.

## CHAPTER I.

### DEFINITION, ETIOLOGY AND PATHOLOGICAL ANATOMY OF REFLEX PARALYSIS.

The generic term *reflex*, used here, comprises that peculiar and extensive class of paralyses called functional, idiopathic, asthenic, and peripheral. It may be proper to state from the beginning that this name does not imply contraction in the blood vessels of the spinal cord as a necessary initial cause of the paralysis, nor subsequent absence of structural change in the nervous system as its effect. I keep the word, because there is no advantage in coining a new one when that already created suffices, provided we define its meaning to guard against looseness of application. Indeed, there should be no objection to call such form of paralysis *reflex*, for several of its varieties attending fever, pneumonia, anæmia, intoxication, etc., could not properly be looked upon as *peripheral*, in the true sense of this expression; while to name them *functional* is obviously in opposition to our knowledge that material modification in the organs is essential to disease. Therefore, as already intimated, disregarding the idea of vascular contraction and absence of lesion in the spinal cord—certainly not conveyed by the term reflex—this name is probably the most appropriate to assign to these varie-

ties of paralysis, inasmuch as their several causes reflect upon the spinal cord, ganglia and nerves, the lesions hereafter to be described.

I will not dwell at any length on the theory which puts forward a contraction of the spinal blood vessels and subsequent diminution of blood, as the original cause of reflex paralysis. Far from my mind is it to doubt the results of the experiments on which Brown-Séquard bases such a theory; but it is also certain that neither the extirpation of the kidney in animals—performed by Le Roy, d'Etiolles, and others—nor the experiments of Gull, Pavy, and Durham, have ever been attended with paraplegia, or the contraction in the blood vessels of the cord, supposed by Brown-Séquard to cause this latter. It is true that, according to the experiments of Kussmaul and Tenner,[1] it cannot be denied that, "anæmia of the spinal cord produces paralysis of the limbs, of the muscles of the trunk, and of respiration. When the anæmia suddenly attains its greatest intensity, then only, and even then but rarely, do slight trembling movements of the limbs precede paralysis." However, in opposition to these facts and his above theory, Brown-Séquard points out that "convulsions take place when the spinal cord is suddenly deprived of blood, particularly if the cord has been separated from the brain by a section at the cervical region."[2] Moreover, Donders and Callenfels have ascertained that irritation of the cervical sympathetic determines contraction, which is immediately followed by a considerable dilatation of the blood vessels. Stilling, Czermak, Bernard, and Schiff have also proved that irritation of the vaso-motory nerves is attended with hyperæmia, otherwise noticed by

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[1] Epileptiform convulsions from hemorrhage. The New Syd. Soc., 1859, p. 105.

[2] Journal de la Physiologie de l'Homme et des Animaux, Tome I., 1858, p. 202.

Simon, Romberg and others as an accompaniment of neuralgia. Finally, Legros, from experiments on the physiology of erectile tissues, communicated to the Biological Society of Paris, October 17, 1865, is led to admit "a sort of antagonism between the action of the sympathetic on the last arterial and venous extremities; arterial dilatation subsequent to contraction of the veins being caused by excitation of the nerve, while its section or paralysis brings about a contrary effect." These are very important facts in relation to the theories on the antagonism between the spinal and vaso-motory nerves, suggested by Henle, Virchow, Simon, and Jaccoud.

It is not my intention to allude here to cases of pseudo-paralysis, so well described by Gull, Spencer Wells, Jaccoud, and, latterly, by Dr. S. Weir Mitchell. Nor shall I engage myself in discussing the no less important question of extreme weakness without original lesion of the nervous system, frequently confounded with paralysis. My principal object is, to point out the histological changes undergone by the nervous system in reflex paralysis, and the facts indicating its relations to the sympathetic system.

The causes capable of giving rise to reflex paralysis are:

Exhaustion of central nervous incitability.

General affections, and a contaminated state of the blood.

Disturbed nutrition by conditions other than the above.

Circumfusa: cold, wet, and atmospheric influences, although these latter more properly belong to those causes acting on the blood.

Lesion of the peripheral nervous system.

It is not proved, as Dr. Mitchell[1] says, that nearly every cited example of reflex paralysis has been free from lesion of the nervous centres, since the statement is not substantiated by any microscopical examination. The instance of paraplegia re-

[1] N. Y. Medical Journal, Vol. II., No. 11.

lated by Dr. Gull[1] in no manner settles the question, for although no spinal alteration was discovered with the microscope, yet the brain, medulla oblongata, and nerves of the affected parts were not examined. The case, accordingly, remains with a dubious significance. The doubt with regard to it is furthermore strengthened, if we take into account that paraplegia may be symptomatic of cerebral lesion. This important phenomenon has not attracted the attention it deserves—its occurrence being frequently denied, notwithstanding its acknowledged existence by Serres, Esquirol, Durand-Fardel, Cheyne, Earle, Watson, Romberg, and several no less reliable authorities. Abercrombie mentions one of the most positive proofs of cerebral paraplegia in a case communicated by Dr. Christison. The patient had intense pain of the left temple, accompanied by deafness of the left ear, squinting of the left eye, and paraplegia. On cadaveric inspection there was found a thickening of the dura mater, with adhesions to the arachnoid on the temporal fossa and above the pars petrosa of the temporal bone, involving the sixth and seventh nerves. There was a tubercle in the right hemisphere, and a small cyst in the right corpus striatum. A portion in the posterior cornu of the right ventricle appeared as if it had been obliterated by adhesion. *No appearance of disease could be detected in the contents of the spinal canal.*[2]

Dr. J. V. Laborde has recorded a case of amaurosis and paralysis of the vagus nerve, with incomplete paraplegia produced by two cysts in the brain.[3]

Dr. Hughlings Jackson[4] has also described several instances of amaurosis and paraplegia, the former being the result of

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[1] Guy's Hospital Reports, Vol. IV., Third Series, Case XVII., p. 174.

[2] Abercrombie, Diseases of the Brain and the Spinal Cord, Phila., 1836, p. 353.

[3] Union Médicale, No. 137, 1859.

[4] "Illustrations of Diseases of the Nervous System," in Clinical Lectures and Reports of the London Hospital. Vol. I., 1864, p. 376.

reflex action set up by the disease of the cord which produces the paraplegia, according to the explanation given by Brown-Séquard. Recently, Dr. J. W. Ogle, in a very interesting paper "On primary Carcinoma of the Brain," p. 4, describes a case in which paraplegic symptoms, loss of memory, difficulty in speech, and dysphagia, existed with a large fungoid growth in the left cerebral hemisphere. Unfortunately, the cord was not examined in this instance. I have lately seen, in consultation with Dr. G. A. Sabine, a girl sixteen years of age who, five years ago, after repeated vomiting and headache, gradually became amaurotic and paraplegic. She is now quite blind. There is atrophy of the optic nerves, enlarged pupils, marked dysphagia, without impediment in speech, and she still complains of the headache. The loss of power in the lower limbs has gradually progressed, with diminished temperature, but without anæsthesia or perceptible muscular atrophy. The arms are free from trouble, with the exception of a slight tingling, occasionally, at the end of the fingers. There is no pain along the spine; the intellectual faculties and general nutrition are failing. We have been led to suspect a cerebral tumor as the cause of the above symptoms.

There is, however, an instance of paralysis from cold and wet in which Dr. C. Hanfield Jones[1] made a microscopical examination of parts of the spinal cord without detecting any trace of organic alteration.

"J. Edwards, aged thirty, young looking, was admitted October 11th, 1859, under Dr. Sibson. Four months ago he was in wet grass all the morning. His legs became stiff the next morning, and his knees weak; he felt as if he were standing on pins. The arms also became affected, but only numb. Subsequently his mastication powers and his voice became impaired; he had double vision for about a month, and after-

[1] British Medical Journal, 1859, p. 309.

wards singing in his ears. On admission, he was observed to lift the jaw with his hand in speaking, and to shoot out his head when he lies down. He walks slowly, lifting his feet. When he gets on his bed he does it in a series of jerks, and then falls flat. Both hands are sensitive. The pupils act. He has vomiting. Pulse 64; respiration 32.

"Oct. 14th.—He complains of pain at the back of the head, and of the arms being more powerless. He can not open and shut his mouth without the help of his hand, when lying in bed. The power of deglutition is impaired. Memory is perfect.

"Oct. 17th.—When his wife came into the ward he went to meet her, pointed to his throat, and nearly fainted. He became excessively pallid; his skin was hot and covered with sweat; pulse, 120. Soon afterwards he sank and died. He had taken iron and quinine, and improved somewhat for a time."

"Post mortem examination: the body was spare, of medium height. The thymus gland was enlarged; it weighed 537 grains. The heart was full of fluid blood, and healthy. The lungs were healthy. The liver, kidneys, and spleen were immensely congested, but firm and quite normal in appearance. The stomach was almost empty. The intestines were empty and contracted. The superficial vessels of the brain were slightly congested. The brain substance was firm; the gray matter was darker than usual; the vascular points were more numerous and evident. The lateral ventricles contained a little serum. The choroid plexus were rather congested. Nothing abnormal was found in the brain; all the parts were carefully examined. The spinal cord appeared to be firmer than usual in the parts corresponding to the lower dorsal and upper lumbar regions; the upper part was much softer than this, but sections of both these portions revealed nothing unnatural in appearance or worthy of remark. Mr. Gascoyne,

who conducted the autopsy, informs me that he considered the softening as a post mortem change. Dr. Sibson and I carefully examined portions of the spinal cord and pons varoli the next day under the microscope, but we could discover no organic morbid change; there was no alterations of the coats of the minute vessels, nor glomeruli. The kidneys were also examined microscopically, and found healthy. The vagi, the fifth pair, the spinal roots, the nerves in the paralyzed limbs, and the ganglionic system were not included in the above examination." This latter circumstance leaves the observation incomplete, as may be shown by the following example.

Leudet[1] reports a case of asphyxia by oxyde of carbon, inducing at first paralysis of the extensor muscles, and afterward complete loss of movement of the right lower limb, soon gaining the left lower limb and the upper extremities. On post mortem examination, the spinal cord and brain appeared undamaged, but there were very evident signs of neuritis in the right sciatic nerve. It will be seen, in the two instances of paralysis from cold and wet (Cases I. and VI.) I shall presently relate, that there were no corpuscles of exudation in the cord, and yet its structure had undergone an alteration, which could not be discovered in many parts by microscopic examination with a low magnifying power. Prior to the late important researches of Lockhart Clarke, the spinal cord had been searched in cases of tetanus without detecting changes in its structure; however, the lesions and alterations of structure are numerous in the organ, but, as shown by the skillful anatomist, appreciable only under glasses of considerable magnifying power. Mr. Lockhart Clarke has, in other instances of paralysis, described degenerations in the columns of the cord, although they appeared healthy under low power.

There still remains another case of general paralysis follow-

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[1] Arch. Gén. de Méd., Tome IX., 1857, p. 476.

ing pneumonia, the sequela of intermittent fever and rheumatism, in which no lesion of the nervous centres was detected with the microscope. Gubler is indebted to Landry for the case which he reports in the Archives Générales de Médecine, 1860, Tome II., p. 718.[1] A man aged 52, previous to his admission into Beaujon Hospital, had pneumonia, and was three times bled, blistered, and kept without food for eighteen days. Emetics were also administered. The patient had a very tedious convalescence, and instead of recovering strength grew weaker and weaker. At length he was seized with formication in the limbs, trunk and face; afterwards the legs became weak and could not support him, nor could he direct their movements, being otherwise incapable of elevating his arms above a horizontal line with the body. Respiration became also laborious, and mastication and deglutition difficult. There was no trembling nor spasms in the limbs, neither loss of control of the sphincters. Muscular irritability and nervous excitability remained undamaged, but the temperature was lowered in the limbs and sensibility deadened in their extremities. There was no fever; pulse 85 to 90, small and soft. He had a cough, with mucous expectoration, and sweating. The paralytic symptoms ran their course in eight days, at the end of which period the patient had constriction of the larynx, with excessive dyspnœa, feebleness of speech, and cyanotic face and neck covered with cold perspiration. At last, after attempting to take some nourishment, he turned pale, collapsed, and died.

On post mortem examination, the rigor mortis was found persisting about forty hours after death (month of June). The sinuses and veins in the cerebro-spinal membranes were filled with blood. No visible or microscopical change was detected

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[1] Des Paralysies dans leurs Rapports avec les Maladies Aigues, etc. See, also, De la Paralysie Amyotrophique consécutive aux Maladies Aigues. Mém. de la Société de Biologie, Tome III., 3ème Série, 1861.

in the nervous centres. All portions were examined with the greatest care, after being prepared in very thin sections. The microscopical investigation was made by Bourguignon, Gubler, Landry, and Robin. The lungs were in a state of splenization and of engorgement, without any tuberculous deposit. The other viscera were not examined. This example, certainly, affords every guaranty as to the non-existence of lesions in the nervous centres, although it is silent as to the condition of the ganglionic system. However, the general asthenic symptoms, or *amyosthenia*, as Gubler calls it, which ended the disease, plainly point out their origin to be the exhausted condition of the patient, from bleeding, blistering, vomiting and insufficient food, and, above all, from the state of the lungs causing incomplete oxydation of the blood, and slow asphyxia. That anæsthesia and paralysis accompany asphyxia is too well established to need further comment. I believe, therefore, that too much value has been given by Gubler to the absence of changes in the nervous centres, and too little to the prominent circumstances in which amyosthenia occurred. Truly, such loss of power constitutes paralysis, but it does not follow from it that it must always have a nervous source, for, in several circumstances, it may be and it is the effect of causes quite foreign to the nervous system; and frequently, again, as in the case before us, it is the lethal sign with diseases deeply affecting nutrition. Leudet[1] reports two cases of paralysis following typhoid fever. In one, the four limbs were attacked; there was asphyxia, and death ensued seven days after the onset of the first paralytic symptoms, on the commencement of convalescence. The brain and spinal cord, carefully examined, did not exhibit any alteration. The brain was slightly marked with red points of hyperæmia. The nerves in the

[1] Remarques sur les Paralysies Essentielles consécutives à la Fièvre Typhoïde. Mém. de la Société de Biologie, Tome II., 3ème Série, 1860, p. 167.

lower part of the abdomen had no morbid appearance, and, as stated by Leudet, the sympathetic was not examined. The second case was one of paralysis with loss of sensibility and movement in the arms, incomplete amaurosis, retention of urine, delirium and death. There was found on the autopsy complete integrity in the brain and spinal cord. From the frequency of local hemorrhages, often intra-muscular, in typhoid fever, and from instances of acute ascending paralysis from blood poisoning, Leudet is inclined, with good reason, to look upon the alteration in the blood as the cause of the amyosthenia following typhoid fever. Let it be added that A. Vogel has described a loss of motory power, due to intra-muscular hemorrhages in the lower extremities, occurring during the convalescence of typhoid fever, and on the first attempts to walk made by the patient. Intra-muscular abscesses may become another source of incapacity to move after typhoid fever, as will be seen in a case to be reported further on.

It is very important to remark, that none of the reports of reflex or peripheral paralysis, without organic lesion of the spinal cord, allude to any microscopical examination of the nervous elements in the organs where the paralysis originated. Consequently, there remains an unexplored field of interesting research, on the morbid changes undergone by the peripheral nerve fibres and cells, in reflex as well as in other paralysis; for in morbid, as in normal physiology, there are local phenomena with the nerves which do not directly originate from the spinal cord or brain, but from their proper automatic action. That peripheral morbid influences may travel through a nerve and injure the spinal cord and brain, is indisputable. Physiology confirms it in the peculiar action of woorara and strychnia on the motory and sensory nerves. The experiments of Bernard and Stannius[1] prove

[1] C. Bernard, Sur les Propriétés des Tissus Vivants, 1866, pp. 339, 341.

that the functions in the nervous centres may be extinguished by the poisonous influence conveyed through the nervous branches. With woorara, the action goes from the extremity of the motory nerve fibre to the motory cell in the spinal cord, but does not reach beyond; while strychnia goes through the sensory fibres, to extend its effects from one to another sensory element in the spinal cord, until it affects the whole sentient system.

Pathology also affords no less valuable evidence of a peripheral lesion traveling backward through the trunk of a nerve to affect the spinal cord or brain—the following being a valuable instance of the phenomenon: A soldier was operated upon for aneurism of the right axillary artery. In applying the ligature, the nervous branches of the brachial plexus detached from the third cervical pair were included; cerebral symptoms followed on the seventh day, and death ensued on the eighth. The post mortem examination showed an abscess in the left posterior cerebral lobe.[1] As evidence, again, of the part which the sympathetic might share in the mode the peripheral derangement exerts its influence, even over organs not directly connected with the injured nerve, we briefly quote a curious case from Le Bret.[2]

A healthy soldier, aged 22, fell during drill, dislocating the right shoulder. The dislocation was reduced immediately, by violent extension. No sooner was the caput humeri replaced in its normal position and the limb left to itself, than the young soldier perceived, without any pain, that paralysis extended all over the arm and forearm. There was total incapacity of movement, with complete anæsthesia from the elbow down to the tip of the fingers, entire loss of motion and tactile sensibility on the corresponding side of the neck and face, with ptosis, and,

[1] Lallemand, Recherches Anatomico-pathologiques sur l'Encéphale, Vol. I., p. 123.

[2] Mémoires de la Sociétié de Biologie, Vol. V., 3ème Série, 1853, p. 119.

from the moment he was injured, the patient remarked that the sight began to diminish gradually and in a perceptible manner in the right eye. When Le Bret saw him, five months afterward, with the above symptoms and ptosis still persisting, the right pupil was contracted and slightly movable. In spite of local blistering and electricity, the paralysis progressed, inducing muscular atrophy in the shoulder, arm and forearm. The fingers, frequently numb, cold, and bluish, were the constant seat of formication and occasional lancinating pain, extending upward to the supra-clavicular space and the cervical region, with great suffering to the patient. A hydropathic treatment improved nutrition and tonicity of the paralyzed muscles and the circulation of the skin, which regained its normal appearance in the hand. The sight and the functions of the eyelid were recovered, as also the power of motion in the face and neck, and of elevation in the shoulder. Sensibility reappeared in the region of the elbow, but no further down. Compression of the deep cervical plexus, and of the origin of the brachial, below and behind the lower insertions of the sterno-mastoideus, at once determined a very acute pain in the whole arm. In reference to the symptoms of the eye, in this case, let me remark that, not long ago, Paget called attention to the change of size of the pupils after injuries of the brachial plexus at the base of the neck. Dr. J. B. Done, formerly House Surgeon in Bellevue Hospital, has communicated to me the following observation, which seems to correspond with the spasms resulting upon irritation of the sympathetic in the neck obtained by Bernard. A man was admitted into the hospital with dislocation of the right shoulder. Previously, he had suffered from dislocation of the left shoulder, reduction of which had been attended with contraction of muscles of the forearm and hand, existing at the time of admission. The dislocation was reduced in the usual manner by gradual extension, during anæsthesia, and was this time again followed by a contraction of the mus-

cles of the right forearm. Both dislocations were the result of accidental injury, and on neither occasion was the state of the pupils noted. While on this subject, it will not be uninteresting to mention that Chassaignac has pointed out a peculiar form of partial infantile paralysis, attacking the upper extremities, and which suddenly occurs upon external injury, chiefly by pulling about the child by the arms. The paralysis is of short duration.

Drs. Mitchell, Moorehouse and Keen, in their last monograph "On Gunshot Wounds and other Injuries of Nerves," report various interesting instances of morbid influence traveling backward, along the injured nerve, to reflect upon the extremities of the main trunk. Duchenne de Boulogne has likewise insisted upon this fact in his work "On Medical Electricity." Brown-Séquard, in his essay "On Diseases of Nerves,"[1] and his lectures "On the Physiology and Pathology of the Central Nervous System," describes the influence of local nervous affections, a subject before studied in an interesting manner by Descot and Bérard.[2] Le Bret refers, in addition to the case already quoted, to three others, reported by Flaubert of Rouen, in which the attempts to reduce dislocation of the shoulder were attended with hemiplegia.[3] One of these cases proved fatal. On post mortem examination, all the nerves of the arm in the axillary region were found united by cellular tissue, which had evidently been the seat of long continued inflammation, and thinned from constant pressure of the head of the humerus. The torn extremities of the four lower pairs of the brachial plexus laid in the tissue of the scaleni, separated from their insertion to the spinal cord. The ganglia of the posterior roots were floating out of the vertebral canal. The peripheral extrem-

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[1] A System of Surgery, by Holmes, Vol. III., p. 877.

[2] Descot, Affections Locales des Nerfs, Paris, 1825.

[3] Répertoire Général d'Anatomie et de Physiologie Pathologique, 1827, Tome III., p. 55.

ities of the nerves looked quite natural. The brain was likewise normal, but the spinal meninges appeared congested and the cord softened in the cervical region. The details of this case may be of interest in regard to the history of the pathology of peripheral paralysis from traumatic causes.

Of such alterations as those undergone by the peripheral nervous system, Longet has been one of the first to describe the condition of the nerves in a case of paralytic varus of the right foot.[1] "The nerves of the right leg were thinner than those of the left extremity. No marked change was perceptible in the spinal cord or the brain; but the anterior roots of the lumbo-sacral nerves forming the right sciatic nerve had scarcely one-quarter the diameter of the corresponding nerve of the left side, while the posterior roots of both sides presented the proper thickness. The atrophied roots had a brown and almost ochrey color. Only one of them escaped atrophy and discoloration." Rokitansky, according to Hasse, has detected spontaneous primitive atrophy of the peripheral nerves, without any well acknowleged cause, and probably due to the influence of cold or violent efforts. H. Mayo[2] says: "What is supposed to be inflammation of the sheaths of nerves is no unfrequent consequence of exposure to cold. I have seen it in the nerves of the arm, and it is perpetually met with in the form of sciatica."

Dr. J. Luys has studied muscular contraction, twice in hemiplegia from apoplexy, and once in paraplegia due to softening of the spinal cord. In all these instances the muscular contraction was induced by paralysis of the antagonistic muscles, upon fatty granular degeneration of the neurilemma, and fatty granular coagulation of the medullary substance, with complete disappearance of the cylinder axis in their nerves. The mus-

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[1] Romberg, "On Diseases of the Nervous System." Sydenham Society, 1853, Vol. II., p. 364.

[2] Outlines of Human Pathology, 1837, p. 143.

eles supplied by these very nerves had also undergone a granular fatty degeneration, and contraction was simply the result of persisting tonicity in the unaffected muscles.[1] I may add that in the same year of 1859 I noted, with Prof. Ch. Robin, a similar condition of the nerves and contracted muscles in cases of Pott's disease, while engaged in my researches on the true nature of the so-called tuberculous affections of the vertebræ. Cornil has examined, also, the nerves of a contracted and paralyzed arm of a woman, who died from an old hemorrhage in the corpus striatum and optic thalamus. The nervous degeneration in this case was mainly caused by hypertrophy of cellular tissue, with hypertrophy and hypergenesis of nuclei in the connective tissue of the nervous sheaths. The nerve fibres were natural.[2]

The most valuable evidence, however, as regards the cause of peripheral paralysis, and accounting for its existence independently of initial lesion of the spinal cord, will be found in the following interesting examples.

L. Dumenil, of Rouen,[3] reports: "In a man of fifty-three, suffering from incomplete motor paralysis of the left arm, there coexisted complete paralysis of the tongue, attended with inability to swallow solid food, and imperfect paralysis of the muscles of the face. The patient having been accidentally killed, both hypoglossi were examined, and found to be highly atrophied and gray in color. The terminal branches in the muscles of the tongue contrasted strikingly with the ramifications of the trifacial and glosso-faryngeal nerves. There was a somewhat similar condition of the peripheral branches of the facial nerve and the anterior roots of the spinal nerves,

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[1] Comptes Rendus et Mémoires de la Société de Biologie, Tome I., 3ème Série, 1859, p. 70.

[2] Comptes Rendus de la Société de Biologie, Tome V., 3ème Série, 1863, p. 8.

[3] A Year Book of Med. and Surgery, Syd. Soc., 1860, and Gaz. Hebd. de Méd. et Chir., 1859, No. 25, p. 390.

especially on the left side. The muscles supplied by the latter were very pale, and far advanced in fatty degeneration. This was not the case with the muscles of the tongue and face."

The same Dr. Dumenil[1] reports another instance of peripheral paralysis of movement and sensibility, with atrophy of the nervous branches in the paralyzed parts. These are the main features of this valuable case: An old man, aged 71, anæmic, thin, and always of regular habits, was at first seized with formication in the left foot and right arm, which, after several days, extended to the left arm. There was no headache or other pain whatever. Subsequently, both feet became completely paralyzed; this was not the case, however, with the legs and thighs, which respectively preserved their movements. There was loss of sensibility to pricking in the lower half both of the external and internal integuments of the right leg, and sensibility was very dull at the sole of the foot. In the left limb sensibility was lost in the external lower fourth of the leg and in the foot, but remained normal in the integuments of the internal region. The upper limbs had only the hands involved; the patient could not grasp at any thing, and the right hand was weaker than the left. The intelligence was clear; no impediment with speech nor inability to swallow; sight, cutaneous sensibility of the trunk, micturition and defecation normal. No pain over the spine, pulse irregular and very unequal, tumultuous beating of the heart, with prolonged first sound, but without venous regurgition or œdema. Electrical irritability was more or less completely lost in the paralyzed muscles of the limbs, and the numbness and prickling never reached above the knees nor beyond the hand.

Cadaveric examination, after death from latent pneumonia: commencing suppuration in the lower lobes of the lungs; slight pleuritic and pericardiac effusion; fibrinous unad-

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[1] Gaz. Hebd., March 25th, 1864, No. 13, p. 203.

hering exudations on the surface of the heart; injection of the visceral parietes of the pericardium, mainly existing over the auricles; volume of the heart normal; left ventricle very firm, round, with thick coats of a dark brown color; aortic valves somewhat hardened, but without insufficiency or stricture; nothing abnormal with the mitral valves. The stomach had a thickening of the mucous and muscular coats around the pyloric orifice. With the exception of old adhesions between the liver and spleen, the abdominal viscera were natural.

The brain, spinal cord and its nervous roots were very carefully examined, and found free from alterations. The muscles, less tinted and developed in the paralyzed parts, did not exhibit any evidences of fatty substitution. The nerves looked natural, excepting, perhaps, the collateral branches of the toes, which were more transparent after long maceration. Dr. Geo. Pouchet, whose great microscopical ability is well acknowledged, examined the nerves and muscles. These latter were not found granular, but they appeared very friable, probably on account of some degeneration undergone by the myolemma, which was not examined. The peripheral nerves in the paralyzed parts and in the sole of the foot—which was the seat of anæsthesia—exhibited a true atrophy of their medullary substance. The lesion involved both muscular and cutaneous branches. The branch of the right median nerve, supplying the muscles attached to the epitrochlea, was found in the early stage of a more distinct and positive alteration than that detected in the plantar nerves. Such degeneration was not discovered in the nerves of parts not paralyzed.

Charcot and Vulpian[1] noticed that, in a case of diphtheritic angina, the muscles of the palate had undergone, in some places, a very fine granular fatty degeneration. The muscular nerves were composed of altered and sound fibres mixed

[1] Comptes Rendus de la Soc. de Biologie, Tome IV., 3ème Série, 1862, p. 173.

together, the former exhibiting a granular alteration, also existing between the fibres and underneath the neurilemma, which contained in addition some granular corpuscles like those peculiar to certain forms of cerebral softening.[1] As to the mucous membrane of the palate and its nerves, they were found natural, with a few rare fatty granulations interspersed in the first.

Finally, in a post mortem examination, reported, without history of the disease, in Virchow's "Archiv. für Pathologische Anatomie," etc., July, 1865, there was found a gray degeneration of the posterior columns of the cord, but the cauda equina and the spinal roots were uninjured, while the sciatic and crural nerves, with their branches, were atrophied, and had undergone a fatty degeneration.[2]

Let us now pass to the results of my own observations, and of four cases very carefully investigated—two by Laborde, and the two others by Cornil and by Kussmaul—and followed by the results of other less complete post mortem examinations, but of important bearing on the subject.

CASE I.—*Paraplegia a Frigore—Meningitic Congestion—No Lesion of the Spinal Cord detected without the Microscope.*—This patient was in the clinic of my friend Dr. T. G. Thomas, at

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[1] Charcot and Vulpian think possible that the altered nerve fibres were motory, and those not altered sensory.

[2] These pages had gone to press when I read in the Gazette Hebd. de Méd. et Chir., Jan. 26th, 1866, No. 4, p. 51, the commencement of Dr. L. Dumenil's "Contributions to the History of Peripheral Paralysis, and especially of Neuritis." The case reported in this part of the paper is, perhaps, the most valuable and complete observation ever published on the subject. I very much regret the impossibility to give a fuller account of it. A healthy woman, upon concussion of the right sciatic nerve, was taken with paralysis of the right leg, inducing eventually a general loss of power and inordinate sensibility, to which asphyxia and death finally ensued. On post mortem examination, a fatty granular degeneration was discovered on the internal sciatic, popliteal, posterior tibial and plantar nerves, in some of the spinal roots, and in the spinal cord, especially, on the gray subtance and posterior cornua.

Bellevue Hospital, in this city. I extract the following details of the case from a very interesting clinical lecture "Upon some of the Diseases of the Spinal Cord," published by Dr. Thomas in the April No. of 1862 of the "American Medical Monthly:"

Agnes Kilvain, native of Ireland; has resided in this country for fourteen years; is married, but has no children; and has been living in a tenement house in this city, performing the duties of housekeeping. Upon inquiry, we found that she has no well marked hereditary predisposition; that her habits have been moderately intemperate, and that she has been a robust woman until this present attack of illness, of which she gives the following history: On the evening of Nov. 23d, 1861—which, she says, was very cold and inclement—she walked out after dark with very thin shoes and stockings, and got her feet wet, and body thoroughly chilled. She retired that night feeling, however, about as well as usual. During the course of the night she awoke from a sound sleep, with a peculiar "heavy feeling in the feet," and finding that she lifted them with difficulty, and that they seemed to be numb and "asleep," she roused her husband, who was by her side, and told him of it. He made light of the symptoms which had "alarmed her," and she in a short time fell asleep again and did not awake until morning; then she found the heavy feeling and inability to move the legs very marked, and getting out of bed with much effort, found that she was unable to stand; in fact, that she was entirely paralyzed in the lower extremities. This took place on the night of the 23d November. On the 6th of December she entered this institution, having been confined to bed, and been paralyzed during the intervening time. We found her now in the following condition: She lies quietly upon her back, and does not appear to be suffering at all; her pulse is 120 to the minute, and in character nearly normal; her skin is warm and dry, but not of a feverish dryness or warmth; her tongue is covered slightly

with a white fur; over the sacrum a slough of considerable size, about equal to that of the extended hand, has occurred. The paralysis of the legs is almost complete—the patient being able only to draw them slowly and with much effort, for six or eight inches, in bed; she is utterly unable to stand, and we may say that the paralysis of the inferior extremities is as complete as it well could be. She complains of nothing beside the loss of power, but has, during her illness, experienced pain in the back. Upon firm pressure a little pain is found to exist over the second and third lumbar vertebræ. Her menstrual flow is irregular. She has occasionally had jerking of the legs, and formication appears to have existed, but not positively.

After very judicious remarks on the symptoms of the case, Dr. Thomas ventures to diagnosticate red softening, at the same time, however, hesitating on account of the great difficulty in determining positively the lesions occurring in the spinal cord. The prognosis was unfavorable. It was ordered that the patient should have her bowels acted upon by a saline cathartic, and that two nitric acid issues should be placed on each side of the spine, opposite the painful part. The patient was kept quiet, and received the best diet afforded by the institution. She went steadily down, growing weaker every day, and died, on the 16th of December, in a state of syncopy.

"*Post-mortem examination*, Dec. 18th. Nothing worthy of note was found in any organ except the cord. This was carefully removed by Dr. Lyman, and as nothing could be learned from an examination by the eye, I submitted it for microscopical examination to Drs. Metcalfe and Echeverria, who made, concerning it, virtually the same report." The results of the investigation I gave to Dr. Thomas in the following brief note:

"The bodies of the four dorsal vertebræ were neither rough

nor changed in their structure; there was no thickening, and very little congestion of the dura mater. The other meninges, however, were much congested, but without any thickening or trace of exudation.

"The external appearance of the cord was natural, and it felt uniformly consistent. Being cut through the posterior median line the gray matter appeared of normal consistency, and exhibited a light rose coloration, more marked in the lower part of the cord.

"In the cervical region (lower part) the gray matter presented two small clots, certainly produced after tearing of the blood vessels during post mortem examination, as there was no congestion around them, nor any of the characters of apoplectic clots.

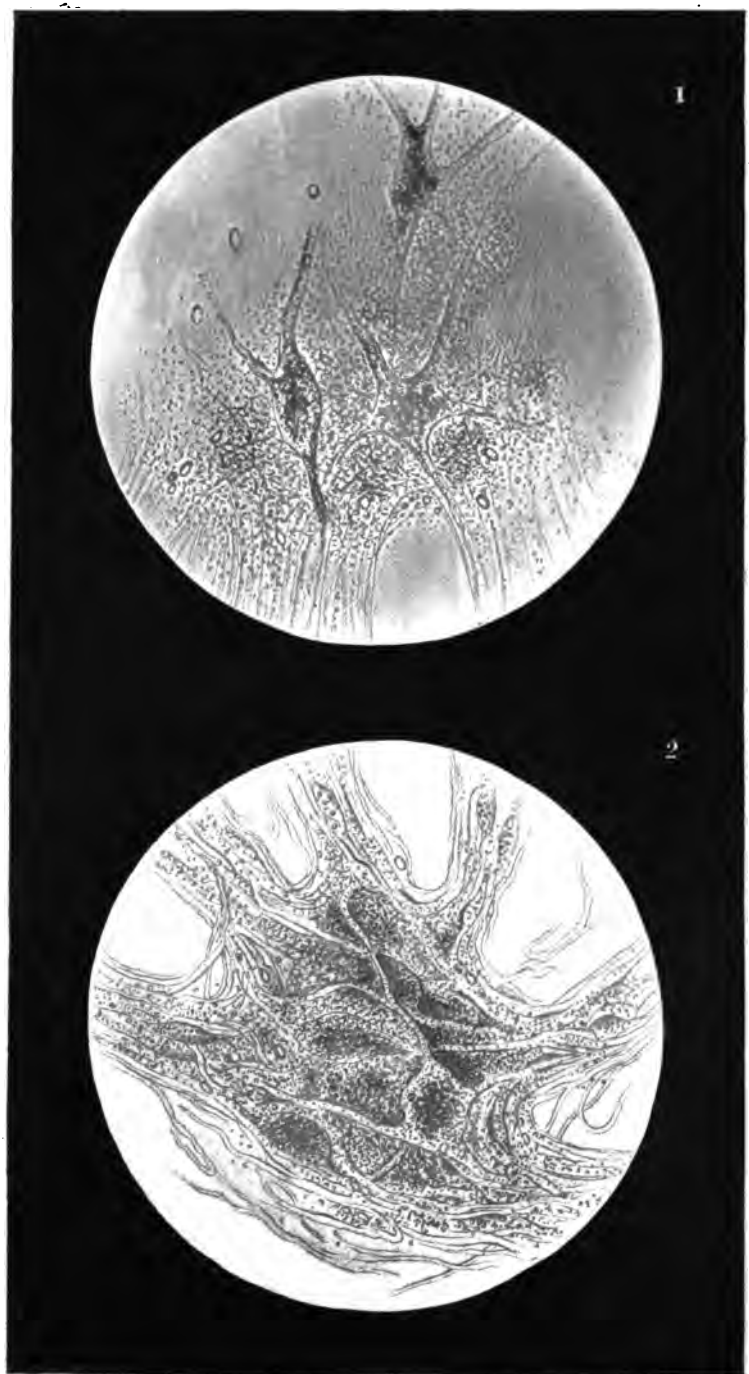
"Examined under the microscope, the white matter was normal, with a congestive state of its capillaries. The gray matter exhibited in the lumbar region a more increased vascularity than in the others. It was found more abundant with myelitic cells and nuclei of connective tissue, and with a more granular amorphous matter; but without corpuscle of exudation or any trace whatever of albuminous exudation; no abnormal existence of fatty cells and granulations. There were, also, some multipolar nervous cells very dark and granular. The ganglia in the lumbar region were abundant in fine fatty granulations, but normal as to the rest of their structure, with the exception of pigment granules abundant in most of the ganglionic cells."

We will state, in addition, that the investigation was made on parts of the cord without previous preparation, and that most of the alterations were manifest only under high magnifying power—eye-piece No. 3 and objective No. 5, Nachet's microscope.

CASE II.—*Infantile Paraplegia—Phthisis—Sclerosis and Amyloid Degeneration of the Cord. Degeneration of the Sym-*

*pathetic Ganglia, and Spinal Accessory Nerve.*—In January, 1863, I saw a boy ten years of age, from the Saint Catherine's House of Mercy, with hemiplegia of the right side and phthisis. The palsy occurred at the age of three, preceded by fever, which lasted two days. Shortly after it the muscles in the neck and the flexors in the forearm became contracted, and wasting gradually progressed in the other muscles, until it reduced the right limbs to nearly skin and bones, with deformities in the joints. The right pupil was larger than the left, though not constantly so. There was no facial paralysis; both cheeks were congested, and the intellectual faculties weak but not impaired. The head was naturally developed. The urine acid, without albumen or sugar, and depositing by heat abundant phosphates. The first signs of phthisis had appeared two years previously. When I saw him, both lungs were the seat of tubercle in different stages of development. He was very weak from diarrhœa and hectic fever; and, without any sign of tuberculous meningitis, died on the 20th of March.

Post mortem examination was made about eight hours after death, weather being quite cold. The rigor mortis existed on the left limbs and the contracted muscles of the neck and forearm, the paralyzed limbs being entirely relaxed. The right pupil was a great deal larger than the left. I only was allowed to examine the spinal cord, portions of which were saved, with the superior cervical ganglion and part of the spinalis accessorius. The ganglion was firmer than natural and of a dark ochrey color, but the nerve exhibited no change from its usual appearance. The cord was laid bare, from the medulla oblongata to the lower portion of the dorsal region. The theca vertebralis readily detached from the vertebral canal, and had no unnatural appearance. On laying bare the cord, the cerebro-spinal fluid was found opaque, with a rose color, and in great quantity. The meninges, very vascular throughout, could be easily detached from the cord. I did

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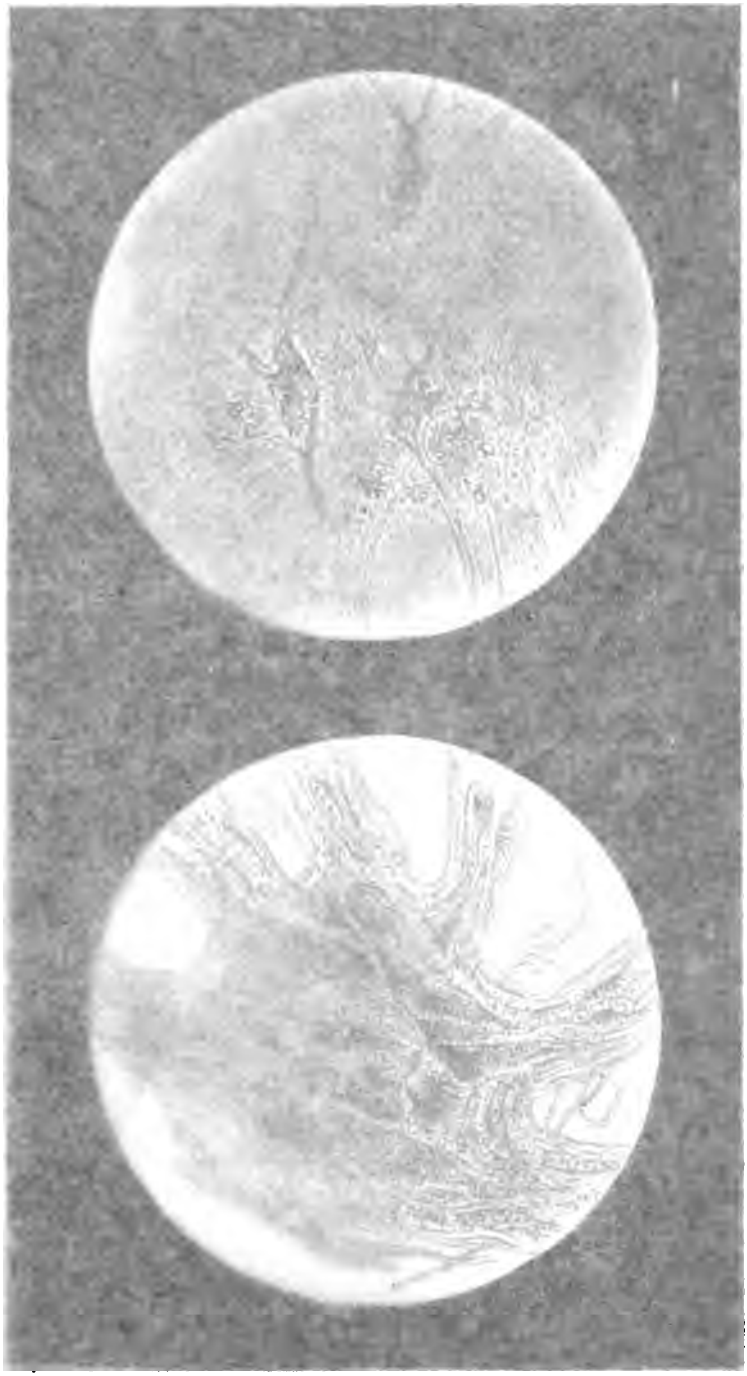

not find, in any place, evidences of tubercular granulations, and did not detect them either over a bare portion of the cerebellum. The spinal cord was softened, particularly on the cervical and dorsal enlargements. Different sections of the cord disclosed no vascular condition of its internal structure; but the surfaces of section were soft and moist, and the gray substance, unsymmetrical in its shape, was, with the white, in many places gelatinous. The more advanced softening was near the anterior median fissure, in the anterior cornua and in the intermedio-lateral tracts.

Under the microscope[1] the gray substance was found much altered in the anterior cornua and intermedio-lateral tracts; it abounded with corpora amylacea and nuclei of connective tissue, and fine granular, amorphous matter of very transparent and delicate appearance. In other places, and especially in the intermedio-lateral tracts, there were groups of brilliant, fatty granulations; undoubtedly, this change was in relation with that undergone by the spinal accessory nerve. The nerve cells had assumed a granular aspect, and were less damaged in the posterior cornua. In the anterior cornua they were filled with pigment granulations, in the midst of very fine fatty granules and amorphous matter. (Fig. 1, Pl. I.) The princi-

[1] In this and the next cases the spinal cord and ganglia were at first hardened in spirit of wine, to obtain thin sections of them. These were macerated in alcohol slightly acidulated with acetic acid, and dipped afterward into pure glycerine until rendered transparent. The use of glycerine, which I have learned since 1856 from my friend and teacher, Prof. Ch. Robin, permits us to obtain a more clearly defined structure of the nervous elements, and, as found by J. Dean, is one of the methods best suited to the minute study of the cord. On some occasions portions of the cord were hardened in a weak solution of chromic acid before putting them up in glycerine. To study the nerves and ganglia, I have used the plan suggested by Liegeois. Maceration of the specimens in tartaric acid renders the cellular tissue quite transparent, thus allowing the number and condition of the primitive nerve-fibres to be easily ascertained.

pal lesion of the white substance existed in the anterior and lateral columns. Besides the corpora amylacea, already noticed, there was a hypergenesis of connective tissue and nuclei, and a fatty granular, amorphous matter surrounding the dissociated nervous elements. The capillary vessels were more manifest near the surface of the white columns; they were not distended, but their coats were finely and brilliantly granular throughout, the alteration appearing to be fatty.

The ganglia in the posterior roots were, like the rest of the cord, covered by the meninges in the above congested condition. Their structure was deeply injured; the nerve fibres and cells had been replaced by a mass of connective tissue and fatty, amorphous matter, with numerous nuclei. Some of the cells contained a great quantity of brown pigment granules. A similar condition, with a greater amount, however, of pigment and fatty granulations, was detected on the cervical sympathetic ganglion, a portion of which is represented, Fig. 2, Pl. I. The anterior roots were much atrophied, with very few granular nerve fibres, and principally constituted by the connective tissue and fine fatty granulations betwixt the primitive fibres. As to the spinal accessory nerve, many of its fibres were reduced to the neurilemma with several nuclei, while others had their natural appearance. The connective tissue and fatty granulations were certainly increased among the elements of the nerve. The capillary vessels in the arachnoid had undergone the same but less advanced alteration described in those of the cord. I found nowhere traces of exudation, and the change undergone by the meningeal blood vessels and by the white columns in some regions was only manifest under very high power.

The fibres of the sterno-mastoideus, contracted during life, exhibited a very fine granular degeneration, which was not fatty. Ether had no effect on the granulations, otherwise rendered transparent with pure acetic acid.

CASE III.—*Infantile Paraplegia—Sclerosis and Amyloid Degeneration of the Cord—Degeneration of the Ganglionic System and Lumbar Nerves.*—A little girl, two years old, became paraplegic after severe diarrhœa and fever, during which she was seized with convulsions. The limbs were very painful from the beginning—the child crying every time it was disturbed—were cold, and commenced wasting immediately upon the appearance of paralysis, which supervened on the diarrhœa. She was again taken with diarrhœa, and became very ill in September, 1864, seven months after the first attack. I saw her two days before dying. She was in a very exhausted condition, with a red tongue coated with aphthæ upon its edges, very thirsty, much troubled with vomiting, and having green, waterish stools, with tenesmus and slight prolapsus recti. The extremities were cold; and from constant vomiting, which prevented her using any remedy or taking nourishment, the child died in a state of stupor on the 28th of September. She had also been troubled with umbilical hernia.

I could only examine the lower portion of the spinal cord, removed from the middle of the dorsal region downward, with part of the lumbar nerves, and the sympathetic ganglia of the inner border of the psoas muscle. In this, as in the former case, the rigor mortis was only present on the upper limbs and upper half of the body, which were the parts not paralyzed. Cadaveric examination was made ten hours after death. Nothing unnatural was noticed in the theca vertebralis; the vascularity seemed increased in the meninges, and their cavity was distended by a whitish thick fluid at the lower end of the cord. The tissue of the cord was softer in the anterior than in the posterior part, and more so in the gray matter. Sections of the cord through the lower dorsal region and the lumbar enlargement revealed atrophy of the anterior and lateral columns. The anterior cornua, the anterior commissure, and the intermedio lateral tracts exhibited to the

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... II., which is ...
... and through the middle
... white substance ...
... power, but magni-
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... opaque degeneration,
... substance. The lesion was
... on the left side, encroached
... columns, and showed very few cor-
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... appearance. The nerve cells
... were filled with brown granula-
... In the most softened portions of
... the intermediate connective tis-
sue was ... fine granular, amorphous matter, and
several ... corpuscles. Fig. 2, Pl. II., shows this change
undergone by the white and ... substances in the right ante-
... cornua. The spinal ... a, not diminished in caliber, was
... the degeneration which existed in different de-
... out the lumbar region to the very filum terminale.
... lumbar nerves were very much shrunken,
... connective tissue with hypergenesis of
... The spinal and sympathetic
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th... ...orpuscles represented in Fig. 2, Pl. II.,
and th... ...interspersed with fatty granules
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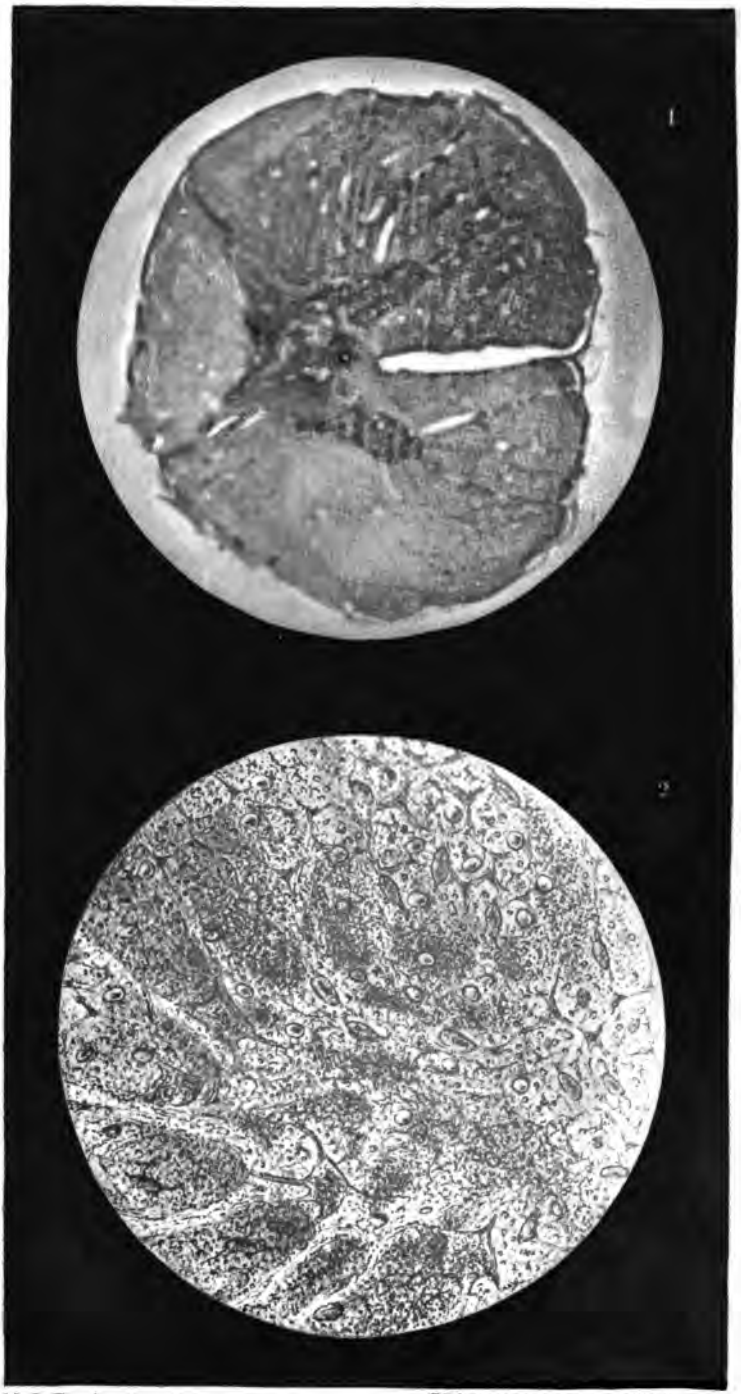

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axis; others had their medullary substance coagulated or granular; they were atrophied in the midst of hypertrophied connective tissue, with several nuclei and fatty granules. The muscular fibres of the psoas presented various degrees of a fatty degeneration.

CASE IV.—*Infantile Paraplegia—Sclerosis of the Cord.*[1]—A girl two years of age was, when eight months old, taken with relapsing fever and general paralysis, which disappeared, leaving her paraplegic. A tonic local treatment improved the condition of the lower limbs, until the girl was seized with cerebral symptoms (vomiting, strabismus, coma), and died.

Post mortem examination: marked meningitic congestion, without plastic exudation; slight serous effusion in the lateral ventricles; tissue of the brain free from tuberculous granulations, or any other structural change. The volume and consistency of the spinal cord was normal throughout. On removing the pia mater, which looked natural, the antero-lateral columns appear as though translucent and of a grayish rose color, instead of having their normal opacity, otherwise preserved by the posterior columns. The same, though less unnatural, discoloration was exhibited by the lateral columns. Several examinations of different regions of the cord, still fresh, showed the lesion involving the whole width of the anterior columns, and only the cortical portion of the lateral. These parts were both superficially and interiorly of a transparent gray color, and less firm than natural. This change was present from the dorsal down to the lumbar region. Under the microscope, the anterior and lateral columns appeared to have increased elements of connective tissue, with cells and nuclei interspersed with fine granular matter composed of fibrils of extreme tenuity. The portions more involved by this morbid tissue hardly contained recognizable nerve fibres, which

[1] Laborde, Paralysie (dite Essentielle) de l'Enfance, 1864, p. 104.

were distended and varicose. There was complete absence of corpora amylacea.

The multipolar cells in the anterior cornua were unchanged, as also the elements in the posterior columns. No structural change was detected in the spinal roots or capillary vessels, nor in the sciatic nerves and paralyzed muscles—these latter having their fibres somewhat shrunken and slightly discolored.

CASE V.—*Infantile Convulsions—Paraplegia—Sclerosis of the Spinal Cord.*[1]—A boy two years of age was, when a year old, taken with fever and repeated convulsive fits, in which he seemed unconscious, and became cyanotic, with evidences of being in great pain. He remained from that time unable to walk, it being impossible to ascertain whether the upper limbs participated in the paralysis at the beginning, although certainly they did not continue long in such condition before recovering their power. Atrophy rapidly set in in the muscles of the lower limbs, causing great distortions of the feet. After entering the Children's Hospital the boy contracted ophthalmia, and afterward measles, with double pneumonia, from which he died the 10th April, 1860. The deformities were greater in the right than in the left limb. Voluntary movement was not abolished, but could only be executed in the direction of the deformities and within very narrow limits. The slightest prickling was acutely perceived, causing pain indicative of exalted cutaneous sensibility. The child was very intelligent; he did not talk, probably on account of extreme weakness, but understood every thing he was told. He could easily sit up; he had no spinal stiffness, nor pain on pressing the spine, and finally no strabismus or sign whatever of cerebral derangement. He wet his bed, it being impossible to ascertain whether unconsciously or from great weakness, and at

[1] Laborde, op. cit., p. 109.

length his life was rapidly extinguished by the double pneumonia.

*Post mortem.*—The muscular contraction and deformities lasted after the rigor mortis disappeared. Neither tubercular granulations nor other lesions were found in the brain—the meninges being slightly congested, and a very small quantity of reddish serosity effused in the lateral ventricles. A thin coat of a somewhat cloudy liquid surrounded the cord within the membranes, forming at the lower extremities a fluctuating tumor the size of a pigeon's egg. This tumor disappeared as soon as the liquid had its exit by puncturing the spinal arachnoid below the lumbar enlargement: the fluid was yellow, cloudy, thick and gelatinous, amounting to about one hundred grammes. The pia mater was of a high red color, rich in vascularity, with whitish frails of milky appearance existing over its surface as far as the sheaths of the nerve roots. On removing the pia mater the cord was firm enough throughout, and unchanged in normal appearance and color. Looking over different sections the neuroglia (connective cellular tissue of the cord) had the gelatinous transparent aspect peculiar to the tissue in children. Nothing, otherwise than the above congestion of the meninges, was manifest in the nervous roots.

The microscopical examination of the cord, still fresh, without any previous hardening, was made on a thin section at the level of the brachial enlargement. It disclosed the following condition: All the elements, and especially the large and small cells in the white and in the prolongations of the gray substances, had preserved their normal state. There was no developement of morbid tissue on the posterior columns and fundamental central substance of the cord. It did not seem, however, to be the same with the anterior columns; their longitudinal fibres appeared in less than normal quantity, and were even absent in some places in the specimen; those persisting were

distended, varicose and fragmented—such condition being principally noticed on the peripheral and quite superficial parts of the said anterior columns. This rarefaction of nerve fibres was detected in no other region of the cord, but all along its peripheral substance the nervous elements had undergone a change in their structure, and were dissociated in fragments, and infiltrated in greater or less abundance with granular corpuscles. Finally, the capillary vessels in the pia mater and peripheral nervous tissue underneath exhibited a greater number of nuclei, and their coats were filled throughout with granules of exudation. This state of the capillaries and nervous tissue existed principally at the lumbar region of the cord, extending, likewise, very evidently to the fibrous sheaths of the spinal roots in the intervertebral foramina. The same, though more distinct, alterations were observed in specimens hardened with bichromate of potash.

It was possible to recognize also the nearly complete integrity of the central structure of the cord, and more particularly of the anterior columns. On comparing the cord with that from a child not affected with paralysis, and examining the more vascular regions, I was scarcely able to appreciate any difference as regards the apparent number of normal elements: that is to say, a slight trace of partial atrophy, and this more particularly on the anterior columns. The difference was, however, so very slight, that I would not venture to affirm that they constituted a morbid condition. Different was it, indeed, with the peripheral regions beneath the pia mater: there the capillaries had considerably increased, and their coats were covered with exudative granulations otherwise infiltrated among the disassociated nervous elements. This lesion was seen throughout the cord from the brachial to the lumbar enlargement, where it reached its maximum. The corpora amylacea were in no place abundant. The same evidences of intense exudative congestion existed in the pia mater,

mainly on its visceral coat. Investigation of the peripheral nervous system only disclosed a relative diminution of nerve fibres in the left sciatic nerve, with abnormal multiplication of fibres of connective tissue. The muscles had undergone different degrees of granular, but not fatty, degeneration, which terminated by reducing the fibres to a hyaline tube constituted by the myolemma.

CASE VI.—*Infantile Paraplegia—Cancerous Cachexia—Sclerosis and Amyloid Degeneration of the Spinal Cord.*[1]—A woman, forty-nine years of age, became paraplegic in 1815, at the age of two, from exposure to dampness and cold. She could not walk until she was eight years old. The greater paralysis was in the muscles of the left foot and leg. In 1860 she had fracture of the tibia, the result of falling, and a second fall during convalescence broke the bone again. The 28th of August, 1862, she was operated on for cancer of the right breast, and six months after the cicatrix and the left breast became painful, with œdema of the right arm. From this date she had chills and fever, with pain along the right intercostal nerves, and dyspnœa, which persisted up to her death, the 10th of October. The lower limbs were atrophied—the feet most completely paralyzed. The left foot was turned in; the right foot and leg were œdematous. Tactile sensibility seemed deadened in this limb, but the other kind of sensibility unaffected.

*Post mortem examination.*—The pericardium, heart, intestines, bladder and uterus, were the only organs not invaded by cancerous granulations. The spinal cord was small, especially in the dorsal and lumbar regions. The antero-lateral columns had undergone a very perceptible atrophy. At the beginning of the lumbar enlargement the antero-posterior diameter was from

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[1] Cornil, Comptes Rendus de la Société de Biologie, 1863, Tome V., 3ème Série, p. 187.

one-quarter to one-fifth smaller than in the normal state. Examined with the microscope the whole length of the cord exhibited, from the first cervical nerves to its very extremity, an anatomical alteration characterized by the presence of a considerable amount of corpora amylacea. These corpuscles were abundant in the anterior cornua, principally in the vicinity of the blood vessels and on the anterior columns. They, however, were also found on the posterior columns. The nerve cells were unaltered, and preserved their normal relations. The left, more than the right sciatic nerve, had undergone atrophy of its medullary contents. Complete fatty substitution, with atrophy of the primitive fibres, had taken place in the muscles.

CASE VII.—*Paraplegia upon Chronic Cystitis—No Lesion of the Spinal Cord—Atrophic Degeneration of the Sciatic Nerve, and Atheromatous Arterial Change in the Abdomen and Lower Limbs.*[1]—A man, æt. 58, entered the Surgical Clinic the 17th December, 1861. He was lame, and attributed it to a false step. When cured of this accident the lower limbs persisted palsied, the paralysis being then referred to chronic cystitis, from which the patient suffered. He had, beside, two large, very painful, scrotal herniæ. No record was kept of the electrical condition of the muscles; but, according to Kussmaul's recollection, they responded pretty readily to the electric stimulus. Extensive mortification set in on the inner region of the left heel, and upon its increased size and suppuration appetite and sleep were lost, and the patient died from marasmus the 22d December, 1862. Autopsy: Body emaciated. Dura mater firmly adhering to the thick, compact calvarium. Sub-arachnoid cellular tissue œdematous. Cerebral convolutions not prominently marked from diminished

[1] A. Kussmaul. "Beiträge zur Anatomie und Pathologie des Harnapparates, Part VI. Zur Lehre von der Paraplegia Urinaria," in Wurzburger Med. Zeitschrift, 1863, p. 56.

depth of the sulci. Substance of the brain pale. Ventricles enlarged, with the ependyma thickened, and a cystic degeneration of the arachnoid plexus. No lesion of the vertebral canal; large quantity of cerebro-spinal fluid in the spinal arachnoid; the tissue of the cord firm throughout, and free from any degeneration whatever, apparent to the naked eye or under the microscope. Slight stenosis of the mitral opening, and also slight hypertrophy of the left ventricle. Intestines displaced on account of the herniæ; peritonitis. Bladder, the size of a goose egg, adhering to intestines; its serous and sub-serous coats were thickened and indurated; the muscular coat, likewise thickened, contained small abscesses, and was separated by exudations from the serous coat. The right kidney, enlarged, presented a general cystic degeneration, which was less advanced on the left. The aorta abdominalis, the iliac, femoral and popliteal arteries were atheromatous, the degeneration reaching its maximum on the hypogastric artery, which exhibited many of its branches changed into a calcareous tube. The inferior vena cava contained a few clots. Microscopical examination of the sciatic nerve showed many of its primitive fibres normal; but in others the medullary substance was coagulated in quadrangular masses, and in others, again, it had undergone a complete degeneration into fatty globules, or aggregates of fine fatty corpuscles. The muscles of the lower extremities had partially undergone also a fatty degeneration. Kussmaul admits that "the paresis of the limbs was caused, in this case, by the degeneration of a portion of the nerve fibres forming the sciatic nerve. Probably the same change was extended to the muscular nerves of the bladder and sphincters, the case being altogether one of peripheral and not central paralysis."

Although no microscopical examination was made in the following cases, they afford, however, manifest evidence of the lesion in the cord. I quote them because, thus far, two of them

seem to have passed unnoticed by those who have inquired into the subject of reflex paralysis.

CASE VIII.—*Petechial Fever—General Paralysis—Suppuration of the Spinal Cord.*[1]—A young soldier, who had lately recovered from a petechial fever, was affected with pain in the dorsal vertebræ, difficulty of moving the lower extremities, retention of urine, involuntary discharge of feces, general debility and emaciation. A variety of treatment was employed for several months without relief. The weakness of the lower extremities increased to perfect paralysis; and, soon after, the superior extremities became affected in the same manner. He then lost his speech, and, after lying a fortnight in this state, completely immovable and speechless, but in possession of his intellectual faculties, he died suddenly.

On inspection, there was found no trace of disease in the brain, the thorax or the abdomen. The spinal canal was inundated by a large quantity of sanious fluid; the cord itself was suppurated, dissolved and disorganized at the lower part of the dorsal region; above this it preserved its figure, but was very soft. Its investing membranes, and the periosteum lining the canal of the vertebræ, were destroyed at the part where the cord was so much diseased; but the vertebræ and their ligaments were sound.

CASE IX.—*Enteritic Paraplegia—Softening of the Spinal Cord.*[2]—A young man, twenty-eight years of age, was admitted into Dr. Serre's ward at La Pitié, the 18th of April, 1825. He had disease of the heart, which was relieved; but symptoms of peritonitis and enteritis ensued. In the course of

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[1] Brera, Della Rachialgite, cenni Patologici. In Atti dell'Accad. Ital., Tomo I., and in Abercrombie, op. cit., p. 295.

[2] V. Robert. De la Paraplégie consécutive à la Fièvre Typhoïde. Thèse. Paris, 1862, p. 33.

this last affection the patient complained of a great weakness in the lower limbs. When the acute symptoms had subsided, and he was thought to have entered upon convalescence, complete paraplegia occurred. The patient could not move the thighs or legs. Sensibility was not lost, but rather seemed to increase along with the loss of power, for the patient cried every time he was moved or touched. The bladder became paralyzed shortly after the onset of paraplegia; there was retention of urine, and on it becoming necessary to keep a sound in the bladder, this organ inflamed. Sloughs soon appeared on the sacrum, and the patient died. On post mortem examination the dura mater was found fungous, the anterior columns softened and disorganized to an extent of three and a half inches, and the posterior columns only slightly altered for about one inch. (Crouzit).

Finally, in a case reported by Fliess,[1] and quoted by Barthez and Rilliet, and by Laborde, the paralysis of one arm was attended with meningitic congestion of the spinal cord, at the level of the brachial plexus corresponding to the paralyzed limbs. No microscopical examination was made of the cord, but it is fair to presume, from what was observed in the above cases, that the meningitic congestion must have coexisted with changes in the apparently normal cord. The same meningitic congestion has been remarked by Briquet in two cases of hysterical paralyis, in which also no microscopical search was made in the nervous centres. Stanley very distinctly notices this congestion in most of his cases of urinary paraplegia. In another instance of paraplegia from wet and cold, Dr. W. Stokes found that the cauda equina appeared to be slightly softened; but from its appearance Dr. Stokes could not state that it was actually diseased. The rest of the spinal cord appeared healthy and normal, without any vascularity, effusion or softening.

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[1] Journal für Kinderkrankeiten, July and August, 1849, p. 39.

External to the sheath of the cord there was a small, flattened, oval body, about the size of half a very small hazel-nut, and of a consistence intermediate between lymph and fat. Around this there was some slight degree of vascularity.[1] Notwithstanding Dr. Stokes' opinion, I think that we are borne out by the details of this case to suppose that there was a change undergone by the spinal cord, which the microscope would have probably detected.

Acknowledging the difficulties in diagnosticating the disease in Case I., I believe it was a typical one of paraplegia upon exposure to cold and wet. Without the microscopical examination made by Prof. Metcalfe and myself, the case would have certainly been added to the extensive number of those in which no visible lesion or exudation has been detected in the spinal cord. Truly, as stated by Dr. Thomas, this could not have been a case of simple congestion, because the attack was too complete and sudden; and if congestion did exist as the pathological state from the beginning, it must have been the forerunner of hemorrhage or softening, for such grave results to have followed. On another hand, the injury sustained by the sympathetic seems evident from the complete paralysis, the fever, and the large slough upon the sacrum, symptoms which rapidly developed themselves, and which could not be otherwise accounted for than by lesion of the vaso-motory nerves. I regret not having had the opportunity of ascertaining the degeneration undergone by the sympathetic ganglia and the muscles. Cases II., III., IV., V. and VI. are examples of functional paralysis, and Case VII., in addition to those of Leudet and Dumenil, pp. 15, 23, proves how the spinal cord may remain unaffected—peripheral degeneration of the nerves accounting for the existence of urinary as well as of any other so-called functional paralysis. I am well aware that it may

[1] Graves, op. cit., p. 359.

be questioned whether Case VIII. was one of true functional paralysis. It is not, however, my purpose to maintain a contrary view. It has mainly been brought out here, because it is one of the rare opportunities in which the fatal termination of the disease has permitted the damage suffered by the nervous system in paralysis consecutive to acute diseases (asthenic palsy of Gubler) to be detected by the naked eye. Case IX. is an interesting example of paraplegia from enteritis.

It may seem, perhaps, strange to find excluded from the preceding Dr. Gull's cases of paraplegia associated with gonorrhœa and stricture of the urethra.[1] They certainly afford the most valuable evidence of the organic lesions which may disturb the nervous functions in the course of urinary affections; but they could not be properly classed with the above cases, and I will briefly adduce the reasons for this opinion. The first case reported by Gull is one of paraplegia following gonorrhœa and syphilis. The paralysis occurred subsequent to the second syphilitic infection of the patient, three days after he had slept in a damp bed, and I would not dispute that this circumstance favored its development; but is it not probable, from the disproportionate relation between the degeneration of the spinal cord—detected with the microscope—and the shortness of the paralysis, that this was rather a direct result of the existing syphilitic affection? The characteristics of the disease and of the spinal alterations described by Gull, the osseous plates on the visceral layers of the membranes, are, indeed, no uncommon phenomena with syphilitic affections of the spinal cord. Therefore, why not consider this as one instance in which exposure to cold, and not the urethral affection, concurred to hasten the progress of the syphilitic change undergone by the spinal cord?

The second case is one of paraplegia, acute spinal arachnitis

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[1] Medico-Chir. Transact., 1856, Vol. XXXIX., p. 195.

and softening of the cord, following retention of urine from stricture. The autopsy was made by Dr. Habershon, and, as acknowledged by Gull, phlebitis, caused by the catheterism, lighted up in higher degree the inflammation existing in the vesical veins in the neighborhood of a pelvic abscess, and which was propagated to the vertebral lumbar region, where one of the veins was found full of well formed pus. The etiology of the paralysis is here very plain, notwithstanding the difficulties of recognizing it prior to the cadaveric examination.

This case is of a quite different nature to that of Kussmaul, Case VII., with which it is associated by Dr. Mitchell,[1] assuredly from want of details in the brief reference made to it by Jaccoud. In such instance, as copied from Kussmaul, there was no lesion of the vertebral canal, and the tissue of the cord, firm throughout, was free from any degeneration whatever apparent to the naked eye or under the microscope. The atheromatous change on the aorta abdominalis, the hypogastric and other arteries of the lower limbs, is not mentioned in connection with the blood vessels of the spinal cord, and, as asserted by Kussmaul himself, the paresis was altogether caused by the degeneration undergone by the sciatic nerve, and probably, also, by the nerves of the sphincters and bladder. It is true, that on alluding to the above *arteritis deformans*, Kussmaul remarks that it may, in an advanced stage, possibly become a source of paraplegia from interrupted nutrition of the lumbar plexus.

The following is the history of the third case related by Dr. Gull in the Medico-Chirurgical Transactions: "Henry F., æt. twenty-one, a pale and delicate man, a shoemaker; habits irregular; has had gonorrhœa many times, and is subject to a permanent gleet, increased when he indulges in drink. His general health has been good, and he was, so far as he knows, quite well on Tuesday morning, March 1st, 1853. In the afternoon

[1] Paralysis from Peripheral Irritation, New York Med. Journ., Vol. II., p. 328.

of that day he began to have pain between the shoulders, and a diarrhœa came on, to which he had been frequently subject. This continued during the night, with increased pain in the back and spasmodic tremblings in the legs. Toward morning the legs became weak and numb, and he was unable to void his urine. His friends, for his relief, applied hot fomentation to the feet, legs and pubes, which produced extensive vesication. He was brought to Guy's Hospital, March 4th, and admitted under the care of Mr. Bransby Cooper, with the following symptoms: complete loss of motion below the sixth dorsal vertebra; the muscles of the seventh intercostal space do not act in respiration; sensation perfect above the line indicated, but on the abdomen pinching or pricking the skin gives no pain, and only the faintest sensation; in the legs there is complete anæsthesia; bladder distended, with dribbling of urine; great exhaustion; pulse 110, weak; respiration tranquil, 24; febrile heat; tongue injected; complains much of thirst; spine quite straight; a slight degree of tenderness and some sense of stiffness between the shoulders. At the epigastrium and about the penis, thighs and ankles, the integuments are vesicated, and the skin is, in parts, sloughing from the hot fomentations which have been applied, and last night, in addition, several bullæ formed spontaneously on the left ankle and on the soles of the feet. No bed-sore; feces healthy, passed involuntarily; urine, drawn by catheter, amnoniacal, and containing mucus and pus, with traces of blood."

"He died from irritation fever and sloughing, March 15th, a fortnight from the beginning of the paraplegic symptoms. Sectio cadaveris: Several superficial sloughs over the legs and abdomen; large sloughing bed-sore over sacrum; bullæ on the soles of the feet; bones and ligaments of the spine healthy. The cord was generally softened as high as the middle of the dorsal region, at which point the nervous substance was broken up by the gentlest stream of water falling on it. The gray

and white portions appeared to be equally affected. Amongst the softened nerve tissue granule cells were abundant. There was no point of suppuration, nor any trace of old disease in the cord. The membranes were apparently healthy; liver healthy; kidneys of a dark color, from venous congestion; the mucous membrane of the pelves slightly ecchymosed; bladder thickened, and the lining membrane covered by recent diphtheritic exudation. Between the bladder and rectum there was an irregular abscess, with sloughing walls, communicating with the bladder by a large perforation of its coats. Near the bulb was a more recent abscess filled with healthy pus. The lungs collapsed freely on opening the chest, and were free from disease; heart healthy."

It appears to me, that sudden occurrence of myelitis (for such, I should judge, was the spinal disease in the above instance) in a patient who had a gleet, but was otherwise quite well, does not distinctly involve a relation between the morbid condition of the spinal cord and urethra. Accordingly, I should hesitate very much to admit such a doubtful causation of the spinal symptoms. Their acute nature and their sudden appearance, without any previous exacerbation of the urethral trouble, are phenomena very much against the assumption; therefore, it would be as gratuitous to make such an affirmation from the mere coincidence or successive development of two morbid states, without marked bearing on each other, as it has been to assure the existence of functional paralysis without any peripheral or central nervous lesion. Dr. Gull, himself, in his valuable paper on Paralysis of the Lower Extremities consequent upon Disease of the Bladder and Kidneys,[1] reports two examples of paraplegia, one with early symptoms simulating incipient phthisis, the other pre-

[1] Guy's Hosp. Rep., Third Series, Vol. VII., 1861. Cases of Paraplegia, pp. 145, 179.

ceded by colics. To these early phenomena, taken individually, might have been erroneously ascribed the cause of the disease, originating in the first case from a tumor in the spinal membranes, and in the second from inflammation and softening of the cord. I remember seeing, in the Charity Hospital of Paris, the next case, whose curious history was communicated to the Biological Society by Dr E. Fournier.[1] The patient, under Dr. Nonat's care, died with incomplete paraplegia, erratic erysipelas, and gradual impairment of the intellectual faculties. He had remained over four months in the hospital, and upon his admission commenced to pass purulent urine. Cauterization of the back relieved him from pain, but electricity applied to the limbs was of no avail to make the incomplete paralysis disappear. The autopsy disclosed: Softening and hyperæmia of the brain; at the origin of the dorsal region a round tumor, the size of a small nut, between the dura and pia mater, loosely adhering to the membranes, and pressing on the cord already atrophied at this level. Kidneys and bladder very much altered; the former enlarged, darker than natural, and much congested; the latter thickened, containing about a glass full of a greenish, pitchy urine, and with a black lining membrane, constricted, dividing the bladder into two nearly equal cavities. No stricture of the urethra.

Jaccoud, noticing the facility with which one is misled in looking for the relation between paraplegia and urinary diseases, refers to the above case of Fournier, and to this other, no less instructive, of Mannkopff.[2] A patient exhibiting symptoms of nephritis and cystitis had pain in the loins, irradiating to the lower limbs, and became paraplegic; the paralysis was

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[1] Mém. de la Société de Biologie, Tome V., 3ème Série, 1858, p. 24.

[2] Les Paraplégies et l'Ataxie du Mouvement, 1864, p. 377; and Mannkopff, Paraplegie bei einem Complicirtem Rückenmarksleiden. Berlin Klinik Wochenschr., 1864.

complete in the right limb, and incomplete in the left. Death occurred at the end of six weeks. On cadaveric examination, it was found: Suppuration of the cervical region of the cord, and of both mediastina, with thickening of the posterior half of the pleuræ; purulent infiltration of the intervertebral foramina, and an inflamed fatty tumor, between the vertebræ and the posterior half of the dura mater, extending some inches down from the lower extremity of the cervical enlargement.

Whether we look upon the described anatomo-pathological evidences as the result of a lesion commencing on the peripheral nervous system, or whether we might again suppose that they at first originated in the muscular or vascular structures, to involve afterward the nervous system, it is undeniable that this latter was the seat of a material change, with common features in most of the cases. We may, thus far, sum up these alterations: a congestive state of the meninges; atrophy and granular degeneration of the anterior and lateral columns of the cord; same degeneration of the anterior cornua of the gray substance, not extending much further than the intermedio-lateral tracts; more or less abundance of corpora amylacea in both substances of the cord, especially with infantile paralysis; granular degeneration of the nerve cells, with hypergenesis of brown pigment granules, mainly in those of the sympathetic ganglia; hypergenesis of nuclei and fibres in the neuroglia and connective tissue of the ganglia; and, finally, a fatty granular degeneration of the peripheral nerves—*neuritis propagata*—capable of being the only lesion accounting for the paralysis.

Dr. Thomas' case is one of the few in which the spinal alteration in paraplegia from wet and cold has been described. This paraplegia *a frigore* should not be confounded with rheumatic paraplegia, from which it differs essentially, as Jaccoud remarks. Its post mortem traces, as far as I have been able to ascertain, are accurately noticed only in five other instances —one of them communicated by Frerichs to Jaccoud, who has

collected three of the examples with which I was acquainted, leaving unnoticed the fourth, reported by Gull.[1] In this case paraplegia came on suddenly after fatigue and exposure to cold, and unattended by any derangement of the general health. There was softening of the cord in the dorsal region; on microscopical examination, the spinal columns were found to be the seat of masses of granular exudations, either free or collected around softened and broken up nerve tubules; there were also granule cells throughout the dorsal and lower part of the cervical region. These cases, though of same etiology, vary in cadaveric appearances from that of Dr. Thomas, reported here, and on this account I have thought better to call separate attention to them. I could not condense the record of the other four better than has been done by Jaccoud,[2] from whom I borrow the following summaries:

A girl, aged 20, noticed a weakness of the lower limbs from the time she had crossed a brook. During three years she exhibited several alternatives of improvement and relapse, the paralysis persisting, however, incomplete and reduced to a mere paresis. At the end of these three years paraplegia ensued, with relaxation of the sphincters ani and vesicæ, paresis of the extensor muscles of the right side of the trunk, with curvature on the left, and finally, bed-sores, hectic fever, general œdema, and death. Cadaveric examination showed: hyperæmia of the pia mater and sclerosis of the spinal cord, the lesion exhibiting itself—quite exceptionally in the longitudinal direction—on isolated patches of induration disseminated along the cord. The patient had had, in addition, cerebral symptoms, and the sclerosis was also detected in one lateral ventricle and the pons varoli.[3]

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[1] Guy's Hospital Reports. Third Series, Vol. II., 1856, p. 168.

[2] Op. cit., pp. 250, 381.

[3] Valentiner, Ueber die Sklerose des Gehirns and Rückenmarks, Deutsche Klinik, 1856.

A man, æt. 51, after exposure to wet, slept a few hours in the open air, with his undried clothes on. Two days after he became paraplegic, and in three days more paralysis assumed an ascending march, death ensuing the twelfth day from the outset of the symptoms. On post mortem examination many softened portions of the cord, with a considerable amount of spinal fluid, were detected.[1]

A man, æt. 37, under the care of Oppolzer,[2] became paraplegic after a fall upon the ice. Post mortem examination disclosed softening of the spinal cord. Microscopic search showed the nervous elements altogether destroyed, mainly on the anterolateral columns. On the most altered space, at the level of the sixth dorsal vertebra, the nerve fibres were undistinguishable, and there was, in addition, a great deal of molecular granulation and fatty globules.

Frerich's case is the following: A child, sitting on a stone for several hours, during very cold weather, was the next day taken with fever, the following with paraplegia, and within a few days more died. On post mortem examination there was found exudative meningitis throughout the vertebral canal.

It is obvious that meningitic congestion and the evidences of myelitis, if such did exist, were by no means as far advanced and unmistakable in Dr. Thomas' case as in those just quoted.

I have found nowhere a description of the change which the sympathetic seems to undergo in reflex paralysis. Rokitansky "has not unfrequently observed a withered state of the ganglia, a shrinking and leather-like toughness of them, while their color has either altogether disappeared or is changed to a rusty brownish, yeast yellow, fawn or slate gray. It appears frequently to be a primary affection; but in many cases it is a

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[1] Walford, Softening of the Spinal Cord, Association Medical Journal, 1854.

[2] Acute Entzündung mit partieller Erweichung des Rückenmarks, Spital's Zeitung, 1860.

secondary consequence of previous disease. The chief example of it is the wasting of the abdominal ganglia which follows typhus, and forms one of the few cases which can be found by the knife of the anatomist for the sickly state succeeding typhus (Typhussiechthum.")[1] To this state of the ganglia might be, perhaps, ascribed the occurrence of paraplegia upon typhoid fever, independently of lesion in the muscular system.

To complete what concerns the nervous alterations in typhoid fever, I will briefly notice, that the nervous tissue may, in addition, undergo chemical changes sufficient to account for the paralysis. Buhl has demonstrated that the proportion of water in the cerebral tissue increases during the commencement and stadium of the fever. Jaccoud thinks, and I fully agree with him, that there is no reason why such œdema should not be present in the tissue of the cord, and become a cause of paralysis following typhoid fever. I believe, furthermore, that a similar œdematous infiltration of the cord, produced by deranged capillary circulation, may possibly be a source of paralysis attending chlorosis and anæmia.

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[1] Rokitansky, Pathological Anatomy, Phil, 1855, p. 344.

## CHAPTER II.

INFLUENCE OF THE SYMPATHETIC SYSTEM ON THE PRODUCTION OF REFLEX PARALYSIS.—INORDINATE SENSIBILITY, CHANGES UNDERGONE BY THE MUSCLES, AND CONDITION OF THE URINE, PECULIAR TO THE DISEASE.

I have had no hesitation, since I commenced to study the pathology of reflex paralysis, in considering it induced through the agency of the sympathetic system. My assumption was based on the intimate connections between the nerves usually affected and the ganglionic system. The spinal origin of the latter and its important influence in the causation of fever and convulsions, often premonitory symptoms of reflex paralysis, most strongly sustain such views. I, therefore, admitted that exhausted incitability of the spinal cord and ganglia were initial phenomena with the disease,[1] at the same time acknowledging, with Brown-Séquard, that vascular spasms of the cord were important elements in the production of the paralysis, and that this was unattended by alterations in the cord. But I accepted the statement with reserve, expecting that the microscope would ere long reveal changes in the structure of the nervous elements better explaining the true nature of this peculiar paralysis. These opinions I have, since 1861, tried to impress on my class, in lecturing on Nervous Diseases, at the University Medical College of New York. The inqui-

[1] Am. Med. Times, 1861, Vol. II., p. 315; Vol. III., p. 36.

ries continued since that date seem to confirm, thus far, the correctness of my expectations.

This theory of abolished power in the nervous centres as a cause of reflex paralysis, was at first conceived by Gull, afterward by Eisenman, 1860, Handfield Jones, *inhibitory paralysis*, 1861, and latterly by Jaccoud, and Drs. Mitchell, Moorehouse and Keen. The last three distinguished physicians suggested[1] that "irritation of the vaso-motory nerves, in a limited part of the spine, might produce contraction of its capillaries, anæmia, nutritive changes, and finally a relaxation of these vessels, which would be more apt to be a lasting condition, and would in fact constitute congestion. Such a series of consequences may very possibly occur, and would no doubt be competent to cause a paralysis, whose site, extent and character would depend upon the part of the nerve centres affected by the excitation." This hypothesis agrees with the one I sustained[2] prior to my ascertaining the cadaveric changes that might attend reflex paralysis, and which still suffices to explain the temporary paralysis consecutive to exhaustion of the incitability of the cerebro-spinal and ganglionic centres, and disappearing on removal of the excitation which produces it. I am in this opinion supported by Jaccoud, who furthermore adduces that the convulsive stage, usually precursory of reflex paralysis, is indicative of exaltation preceding the exhaustion of nervous force. The following cases, observed by Nonat and myself, afford strong evidence in favor of these views.

A young, unmarried lady, aged 22, was affected with metritis, intra-uterine granulations, spasmodic contraction of the cervix uteri, vaginitis, and peri-uterine abscess, attended with general nervous symptoms. She underwent a lengthy antiphlogistic treatment, and several uterine cauterizations. The first appli-

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[1] Circular No. 6, Reflex Paralysis, March 10, 1864, pp. 16, 17.

[2] Am. Med. Times, Vol. III., p. 36.

cation of nitrate of silver to the cervical canal was followed by complete anæsthesia in the lower extremities. Afterward, every intra-uterine cauterization produced loss of consciousness, with momentary paralysis of the lower limbs, and on one occasion the right leg remained as though paralyzed, for several weeks after the operation.[1]

A lady, after a third miscarriage, remained with enlarged uterus, extensive ulceration of the cervix, and metrorrhagia. A mild induced electric current, applied to the cervix uteri, awakened contraction of the organ and stopped the hemorrhage; but was immediately attended with severe pain in the pelvis and lower extremities, tremor and numbness in the limbs, general perspiration, and the secretion of milk, having stopped for five days, was again re-established. Pain subsided on discontinuing the application of electricity; but the lower limbs remained paralyzed, and did not recover completely their functions until fourteen hours after the above operation.[2]

These instances are positive proofs of the above statements. Jaccoud, on quoting the second case, very justly remarks that it seems to be a reproduction of the paralyzing effects which a constant electric current is capable of determining when applied to the spinal cord and nerves, as demonstrated by the experiments

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[1] Nonat, Maladies de l'Uterus, 1860, Obs. LXX., p. 830.

[2] Am. Med. Times, 1863, Vol. VI., p. 2.

Note.—A case of hemiplegia following shocks of electricity, with persistence of the paralysis, has been recorded by Dr. O'Connor, of the Free Hospital. A girl, aged fifteen years, exhibited the paralytic symptoms about seven weeks after she was electrified. She had never menstruated. A fit of about an hour's duration, dysphagia, difficult articulation, unconsciousness, etc., supervened in the course of the paralysis; and although these symptoms yielded to the treatment instituted by Dr. O'Connor, it is not very evident that they were a direct result from such a distant cause as the electric shock, and were, most probably, due to some amount of hysteria present, as suspected by Dr. O'Connor.—*Lancet*, July 27, 1861, p. 87.

of Valentiner, Matteuci, Du Bois Reymond, Weber, Riter, Pfluger, Remak, and Baierlacher. Therefore, always acknowledging exhaustion among the causes of reflex paralysis, and without denying the contraction of the blood vessels of the cord, which I, however, consider a secondary element, it is proved that a material alteration undergone by the spinal cord and ganglia, as well as by the nerves and muscles, in fatal cases of reflex paralysis, has, on closer examination, been detected. I believe, furthermore, that there are cases, such as those of progressive locomotor ataxy, progressive muscular atrophy, etc., which will probably be included in the same class of paralysis due to original lesions of the sympathetic. This classification will be possible when we become more familiar with their etiology, and the morbid changes they occasion in the sympathetic system. By way of proofs to this opinion, I will briefly point out the following facts:

Dr. Baerwinkel, of Leipsic,[1] states that in progressive muscular atrophy the first trouble originates with the sympathetic to involve afterward muscular nutrition and cause paralysis. Muscular inaction brings on degeneration of the motory nerves and softening of the spinal cord takes place by propagation of the morbid process to the sympathetic fibres destined to the nutrition of the cord. As to the origin of the disease, it is to be detected probably on the spinal ganglia. These statements are based on very accurate observation of cases of progressive muscular atrophy. A peculiar degeneration of the spinal ganglia has been noticed, by J. Luys, in progressive locomotor ataxy.[2] Charcot has also detected, after this disease, a great quantity of pigment in the cells of the ganglia in the posterior spinal roots—a condition which he, however, does not

[1] Vierte Gahrschrift für die Praktische Heilkunde, Vol. LIX., 1858.

[2] Bourdon, Études Cliniques et Histologiques sur l'Ataxie Locomotrice Progressive. Archives Générales de Médecine, 1861, Tome I.

consider as a true morbid alteration.[1] Duchenne de Boulogne has tried to prove that the sympathetic is affected in locomtor ataxy.[2] Donnezan[3] has found, in this affection, the cervical ganglion of the sympathetic hard and tough, and with a peculiar yellowish white tint, instead of being as normally of a pink color and of elastic consistence. There was no microscopical difference between this and a healthy ganglion, but in the ramus communicans lamellar tissue had replaced the nervous substance, giving it the appearance of a tendinous tissue, and the nervous elements had mostly disappeared, those few left being in an altered condition. A. Voisin[4] has remarked that with progressive locomotor ataxy dilatation of the pupils immediately follows upon pinching, pricking, or irritation by galvanism of the trunk or limbs.[5]

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[1] Comptes Rendus de la Société de Biologie, Tome IV., 4ème, Série, 1862, p. 171.

[2] Recherches Cliniques sur l'Etat Pathologique du Grand Sympatique dans l'Ataxie Locomotrice. Gazette Hebdom. de Méd. et Chir., Nos. 8 et 10, 1864.

[3] Gazette Hebdom. de Méd. et Chir., No. 19, p. 309, 1864.

[4] Ibid. Journal, 1864, No. 38, p. 633.

[5] After these pages were written, I have seen a case of progressive locomotor ataxy, in consultation with Dr. L. A. Sayre. The patient, a gentleman about 40, has been for two years affected with the disease. Whenever the legs or arms are pinched or pricked, the pupil in the corresponding side dilates. The same irritation, or any effort exerted by the patient, is followed by perspiration, but only on the palm of the hands and on the fingers. At the commencement of the disease the patient had numbness and loss of sensibility in the legs. At that time he could not walk in the dark, nor close his eyes, without falling; now, the anæsthesia has disappeared from the legs, still subject to the sharp shooting pain present from the beginning, and he can move in the dark and keep his eyes closed without falling. Dr. Sayre ascertained the existence of the former facts, and the patient describes the latter as the only sign of improvement he remarks in his disease. These symptoms also bear strong evidence to my statements on page 74. The patient complains that his tongue places itself sideways and is bitten during mastication, and that he has lost gustatórial perception. The symptom with the tongue has

Bazire[1] reports one case of locomotor ataxy in the National Hospital for Epileptics and Paralytics, under the care of Dr. Ramskill. Whenever the patient was seized with paroxysms of pain in the limbs, the left side of his forehead was covered with large drops of perspiration, whereas the corresponding portion of the face, on the right side, remained remarkably dry. The left pupil, however, which was always larger than the right (and strangely enough, for there was partial ptosis on that side), did not dilate any further during the paroxysm. Finally, the late Remak has successfully employed in locomotor ataxy a constant galvanic current applied to the sympathetic ganglia. This eminent physiologist, in his experiments at the Charity Hospital in Paris, demonstrated that the superior cervical ganglion is the point of a centrifugal excitation producing *diplegic* contractions, *i. e.*, reflex movements in the muscles, set up by simultaneous excitation of two distant ganglia, the positive pole being always applied to the cervical zone.[2]

I am not the first to bring to light this influence of the sympathetic on reflex paralysis. Morgagni, Camper, Portal, Boisseau, and recently Romberg, Grisolle, and Axenfeld, assert that the origin of functional or sympathetic paralysis is to be found in the ganglionic system. Romberg[3] says, "As yet the reflex neuroses, for which the sympathetic and its ganglia offer the most productive soil, have not in any way been cleared up by the scalpel or microscope, even as they occur in the brain and spinal cord." We would also remark, in relation to paraplegia from renal disease, that W. Hinds[4] has ad-

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been observed by Topinard in one case; I have met with it in another instance of locomotor ataxy, and my friend Dr. M. Clymer tells me that he has found it one of the earliest unnoticed symptoms of the disease.

[1] Transl. of Trousseau's Clinical Medicine, p. 189.

[2] Remak, Application du Courant Constant, Paris, 1865, p. 28.

[3] Op. cit., p. 336.

[4] British Medical Journal, April 9th, 1859, p. 290.

vanced the opinion that the paralysis is produced by the extension of the renal disease to the lumbar plexus, and admits that its close proximity to the kidneys allows the morbid process to extend from one to the other. In 1857, Spencer Wells,[1] in relation to the same subject of urinary paraplegia, made in one of his lectures the following statements: "If you disturb the normal condition of the sympathetic nerves supplying the kidneys and bladder, you need not be surprised at an alteration in the condition of the spinal nerves with which the disordered sympathetic nerves communicate, or in the conditions of the muscles which derive their motor power from the spinal nerves, whose relations with the sympathetic have been disturbed." . . . . "Another reason for supposing that the sympathetic nerve plays an important part in this form of paraplegia is that gastric derangement is generally a very early symptom. . . . . Admitting the influence of the sympathetic, you can explain them readily enough by the intimate connection of the renal and solar plexuses, without bringing chemistry to you aid."

I have already cited a case from Le Bret, which prominently shows the relation between the sympathetic and reflex paralysis. I take from Gubler[2] the following no less important one observed by Pidoux, at Lariboisière Hospital. A man, aged 52, and in very unfavorable hygienic conditions, was affected with pneumonia on the left side. Cupping, blistering and kermes, with quinine, were resorted to, and the patient entered upon convalesence on the eleventh day. From this date he had mydriasis of the left eye, with ptosis, and, in a few days after, paralysis of the tongue and pharynx. There was no evidence of intra-cranial exostosis, or syphilitic fibro-plastic

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[1] British Medical Journal, 1827, p. 495.

[2] Archives Générales de Médecine, 1860, Tome II. p. 718.

exudation at the base of the brain. Subsequently the fingers became numb, with difficulty of executing any delicate movement or grasping. Eight days after the onset of these last symptoms there were paleness and coldness of the feet, with insensibility of the sole, difficult gait, staggering, without paralysis of the pelvic viscera. Stimulant frictions, nux vomica, sulphur baths, quinine, aloes, etc., good food and coffee, brought about diminution of the paralysis with complete recovery, in about six weeks from the initial accidents attending the convalescence.

Gubler, guided by his former researches on the redness and heat of the cheeks as a sign of pulmonary inflammation,[1] explains the paralysis of the tongue and pharynx in the above case, by *depression* conveyed, in a reflex manner, through the nerves of the respiratory apparatus to the part involved. He is, moreover, inclined to give such sympathetic character to the phenomenon, on account of the exceptional early loss of sensibility and motory power in the arm, which necessarily makes us look for their cause in conditions of close organic proximity. I would rather accept another view suggested by Gubler, and which would explain the redness and increased temperature of the cheeks by paralysis of the sympathetic, capable otherwise of bringing about the other noticed symptoms. The paleness and coldness in the limbs, their insensibility, and the mydriasis, are phenomena, however, very much opposed to this latter opinion, inasmuch as they are consecutive to irritation of the sympathetic, capable of being, in addition, attended with paralysis as well as with hyperæmia. Let me state, that Gubler also admits the possibility of peripheral paralysis being produced by the inflammatory process extending to the neighboring nerves. The existence of paralysis in the throat and arm prior to that of the lower limbs may be readily accounted for, remembering that sympathetic ganglia are centres of reflex

[1] Union Médicale, Avril et Mai, 1857.

actions, always localized in the regions where they originate, and incapable of being general over the organism, or of having a decussated effect, unless they pass through the spinal cord. Another cardinal fact is, that these very local reflex actions, having their centre in the ganglia, are set up, not after special irritations, but after those equally acting on the nerves generally, the former determining their effects exclusively on the cerebro-spinal system. Bernard, who has established these fundamental principles, has proved, beside, that there is a paralyzing reflex agency peculiar to the sympathetic.[1] On accepting this explanation, I am far from excluding the latter one admitted by Gubler, since, undoubtedly, neuritis is a frequent cause of peripheral paralysis. Another important cause of amyosthenia, which Gubler puts forward on explaining paralysis from pneumonia, is the want of hematic oxydation due to the lesion suffered by the respiratory apparatus. The assumption is moreover sustained by similar effects observed in asphyxia. Richardson[2] has established, taking the suggestion from Snow, that suspended oxydation in the tissues is a necessary requisite for anæsthesia. As Richardson lays it down, this arrest of oxydation means, in the end, arrest of motion, and no doubt this inertia in the molecules of the organ, whatever be its origin, is the principal element in paralysis generally. Apart, however, from poisoning, which may at once arrest the nervous functions, or from inflammation, the peripheral nervous changes may be also induced by a deranged nutrition supervening upon a contaminated state of the blood, as in diphtheria, typhoid fever, rheumatism, etc. The sympathetic, in these cases, may become the source of altered activity in the muscles, either operating directly on them or through the blood, modified in its quality. I shall show, hereafter, that the derangement

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[1] C. Bernard, op. cit., pp. 344, 388, 390.

[2] Med. Times and Gazette, Feb. 3, 1866, p. 115.

of the sentient and motory nervous system, following disturbed action of the sympathetic, is of common instead of rare occurrence in reflex paralysis. As to the temperature of the paralyzed limbs, it has been noted by accurate observers to be more or less diminished, and I have very frequently found it to be so. It is not, again, a rash inference from the peculiar migration of the paralysis from one to another organ, and from the variable degree in which the parts are respectively involved, to think that the ganglionic system is and may solely continue affected until the lesion reaches the spinal cord. This view would explain why, in cases of local paralysis, one set of muscles is only damaged, while others supplied by branches of the same nervous trunk remain unaffected. I am not the only one, indeed, disposed to contend that the nervous derangements which are chiefly seated in the peripheral system during the course of typhoid and other fevers, diphtheria, etc., have this ganglionic source. Fritz,[1] who has made a valuable inquiry into the spinal symptoms of typhoid fever, considers the above phenomena as independent of any cerebro-spinal influence. He says that Griesinger, being attacked with typhoid fever, observed from the commencement a loss of sensibility in the mucous membrane of his mouth, without any other alteration whatever. Arthritic pains are noted by Gendrin, and Poulet has, during an epidemic of typhoid fever, observed that odontalgia, otalgia, and pleurodynia existed prior to any other symptom of the fever. I have already noticed the ganglionic degeneration described by Rokitansky in cases of typhoid fever, a fact not to be disregarded in connection with the views here sustained.

I have alluded to reflex actions pertaining to the ganglionic system, which Fletcher considers as the source of all irri-

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[1] E. Fritz, Études Clinique sur divers Symptoms Spinaux observés dans la Fièvre Typhoïde, 1864, p. 69.

tability in the body, but there is additional evidence of the automatic power of the ganglia to be centres of movements, independently of the spinal cord. Graves[1] was one of the first to call attention to a symptom occasionally observed in reflex paraplegia. "The patient," says this author, "complains of tenesmus, and thinks he is about having an attack of piles." "The same observations apply to the bladder, with this exception, that the morbid irritability of this organ occurs occasionally after the disease is confirmed and has made considerable progress." It is, indeed, curious to find that section of the lumbar nerves, or of the pneumogastric, is followed by identical phenomena as those pointed out by Graves in the sphincters ani and vesicæ, and in the cardiac orifice of the stomach. The persistence of such singular contraction, often prolonged for a considerable time, is due, according to Bernard, to the influence of the numerous ganglia in the vicinity of those organs.[2] Ollier, d'Orleans,[3] examined a monster fœtus anencephalous, in which the spine was open from the base of the skull to the renal region. The spinal cord was lying underneath a transparent membrane; it began in a bifid extremity at the base of the cranium, and had a ribbon form about one and a half lines thick. This cord had no communication whatever with the nerves: they had, respectively, their origin in a ganglionic extremity, situated in the intervertebral foramina. During pregnancy the movements of the fœtus could be distinctly detected by palpation, and when born the monster remained motionless, notwithstanding the care taken with it. Instances are reported by Fauvel and Méry, in which the anencephalous fœtus lived some hours, and Lallemand mentions a case in which the movements of an anencephalic fœtus were all the time felt by the

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[1] System of Medicine, Phil., 1848, p. 362.

[2] Op. cit., p. 380.

[3] Comptes Rendus de la Soc. de Biologie, Tome II., 1ère Série, 1850, p. 106.

mother. On the other hand, Velpeau[1] has presented several examples of destruction of the cord with preservation of movement in the limbs, reported by Ollivier, Rullier, Bayle, etc., and since that time other similar cases have been recorded. Finally, a parasitic monster has been described by Chauveau.[2] It was mostly constituted of the posterior limbs, and in one of them only the sciatic nerve existed, connected with a lateral mass of conglomerated spinal ganglia, thus evincing their capital influence on the functions of the peripheral nerves, and their trophic power discovered by Waller.

Romberg,[3] alluding to the influence exerted by the sympathetic upon the contractile elements, says: "In this respect, the skin demands an especial consideration; and it is, indeed, strange that the veterinary surgeon pays more attention to its vigor or tone in animals, than the physician does in diseases of human beings. In intestinal gangrene, in which there is an undoubted paralysis of the sympathetic, we find the skin presenting a similar condition as in Asiatic cholera."

Sir Henry Holland,[4] after presuming that the ganglionic nerves are those which chiefly give their appropriate irritation to the blood vessels throughout the body, says: "We have often local changes in circulation, rendered very singular by the preciseness of their limitation to certain parts, in accordance with the distribution of particular nerves. This is well seen in certain slight and partial paralytic affections, where a portion of a limb, or even one or two fingers, may become bloodless, while the others retain their natural state. I have also seen cases where the perspiration of a palsied limb was singularly altered in the quality of the matter perspired." This

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[1] Archiv. Gén. de Médecine, 1825, Tome VII., p. 329.

[2] Journal de la Physiologie de l'Homme, 1863, Tome VI., pp. 346 and 361.

[3] Op. cit., p. 338.

[4] Medical Notes and Reflections, 3d ed., 1855, p. 135.

last phenomenon has been noted as an accompaniment of epilepsy. The case of locomotor ataxy, quoted from Bazire, is another curious instance of unnatural perspiration. Abercrombie[1] says that "a child, mentioned by Dr. Falconer, became pale and emaciated on the whole left side of the body, without any diminution of muscular power, the right side remaining healthy. She recovered by the use of warm pumping." Travers[2] calls *prostration without reaction* the paralyzing influence transferred from cerebro-spinal to sympathetic nerves and ganglia. Failure and disappearance of the pulse, weakness and fluttering of the heart, coldness and lividity of the cheeks and lips, apathy, unembarrassed consciousness, sopor, with difficult respiration, relaxation of the sphincter, and convulsions—such are the main symptoms of this state.

I am attending, with Dr. L. A. Sayre, a boy with infantile paralysis, supervening upon fever and gastric derangement. The left limbs are paralyzed. When I first saw him, the leg was very much atrophied, cold, and very sensitive, especially near the joints. The left pupil was larger than the right; capillary circulation in the cheek and ear of the same side was very irregular, the skin of those parts presenting congested patches, with more or less dilatation of the left pupil. In addition to these symptoms, from the commencement of the disease the boy has been troubled with epistaxis, difficult to stop. The galvanic excitation of the lower limb, or the hypodermic injection of strychnine in it, has been attended with immediate dilatation of the left pupil, greater congestion, with increased temperature of the face, dizziness, and perspiration in the left hand. These phenomena were very perceptible at the beginning of the treatment, and have now

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[1] Abercrombie on the Brain and the Spinal Cord, Phil., 1836, p. 236.

[2] An Inquiry concerning that Disturbed State of the Vital Functions usually denominated Constitutional Irritation, London, 1827, p. 106.

subsided with the improved condition of the limbs—their intensity, however, being in relation with the alternatives shown by the paralytic state. In all the cases epistaxis has been preceded by redness of the left ear and cheek.

Velpeau[1] describes the case of a very robust soldier, in the Hospital of Tours, who had insensibility and complete loss of voluntary power of the right arm, from the shoulder to the middle of the forearm. The hand was not in the least affected, the skin of the insensible parts was livid or bluish, like that of the face in persons suffering from aneurism of the right heart. This man never had pain in the back, and passed unaware into this condition, without any appreciable cause. Frictions with tincture of cantharides and other excitants proved useless, until, tired of having remained three months in the hospital, he left it without experiencing any change in his disease. However, three months after, the limb had insensibly recovered its functions, without any remedy.

Stronger evidences of the irregularities of circulation in relation to paralysis, and mainly due to lesion of the sympathetic, are the following interesting examples, taken from Abercrombie.[2] "A lady, mentioned by Dr. Storer, was recovering from a pneumonic attack, when one morning, after a restless night, she was suddenly seized with an acute pain in the left shoulder, extending to the arm, and at the same time the whole left side became paralytic. The leg retained an obscure degree of motion and feeling, but the hand and foot were insensible to the prick of a needle. The parts were cold, and all the arteries in them were without pulsation. On the right side of the body the pulse was of a good strength, and a little frequent. After a few hours the pain shifted to the leg and foot; and she had also some obscure pain in the forehead, which was removed by

[1] Sur une Altération Profonde de la Moelle Allongée, etc., Arch. Gén. de Méd., 1825, Tome VII., p. 81.

[2] Op. cit., p. 233.

bleeding with leeches. The pain of the leg and foot abated after twelve hours, and she had then no complaint except the paralysis. For several days she seemed to be improving a little in the motions of the parts, but they continued cold and without pulse; on the fifth day, she had an uneasy feeling in the epigastrium, with sense of suffocation; her breathing became short and hurried, and she died in the night. A gentleman, mentioned also by Storer, was seized with paralysis of the right arm as he sat at breakfast, having been previously in perfect health. He did not complain of any pain, but the arm was pale, and every part of it without pulse; in the left arm the pulse was natural. After four hours he became faint, with quick and laborious breathing, and frequent pulse, and in two hours more he died. The body was not examined." In the Transactions of a Society for the Improvement of Medical and Surgical Knowledge, Vol. III., from which these cases are copied by Abercrombie, Dr. Wells has described that of "a gentleman subject to cough and dyspnœa, who awoke one morning with a severe pain in the left arm; in the afternoon it became benumbed and paralytic. The pain then ceased, and the arm was found to be without pulse. He continued in this state for two days, without any other complaint, and on the third day he died suddenly, as he got up to go to stool. The paralytic arm only was examined after death, and in it no morbid appearance could be detected." By way of contrast with these cases, I will briefly mention the most important details of a remarkable one reported by Sir Henry Holland.[1] "A boy, four years old, had been bled to the extent of nearly 30 ounces. He was delirious; the pulse 120 to 136, hard, and with a tense vibrating thrill in all the arteries, especially those of the head—which I do not remember equally to have noticed in any other case—and felt in the smallest artery to which

[1] Op. cit., p. 137.

touch could be applied. Nearly six weeks from the earliest date of the illness dropsical symptoms came on and rapidly increased. All the preceding symptoms, and notably those of the cerebral irritation and disordered action of the arteries, abated in the same ratio as these dropsical symptoms came on."

It may be supposed that the cases cited from Abercrombie were due to arterial embolism. Such an idea could not be entertained, especially as regards the first case. Hemiplegia, upon cerebral embolism, is usually attended with facial paralysis, and other cerebral symptoms absent in this instance; the accident is never attended with complete anæsthesia before gangrene sets in in the limbs; their sensibility and temperature are increased instead of being diminished, and with these symptoms the limbs exhibit, in addition, more or less signs of partial mortification, from arrested circulation. These are, at least, the results established by E. Lanceraux, in his valuable monograph "Sur la Trombose et l'Embolie Cérébrales." (Paris, 1862.)

It seems to be the prevailing opinion that with reflex paralysis sensibility is not to any great degree impaired. Brown-Séquard[1] says that in reflex paraplegia there is "no formication, no pricking, no disagreable sensation of cold or heat in the affected limbs"—"anæsthesia being also rare." The contrary symptoms characterize paraplegia from myelitis, and Brown-Séquard draws the distinction between both affections taking urinary paraplegia as typical of the reflex variety. Such assertions as regards the state of sensibility in reflex paralysis are, indeed, far from being controlled by experience; nay, they are in opposition to the statements made by those authors who first called attention to this peculiar form of paralysis. Thus, we see that Fabritius, on speaking of dysenteric paralysis, says that patients "are at the same time seized with immobility of one arm and the foot of the opposite side; with this symptom

[1] Paralysis of the Lower Extremities. Phil., 1861, p. 33.

violent arthritic pains are occasionally associated, but most frequently these patients lose all sensation." Romberg,[1] from whose excellent work we make this quotation, says that "anæsthesia is rarely present in the reflex paralysis arising from intestinal affections." He, moreover, establishes that the paralytic condition is occasionally associated with pain, and commonly with atrophy.[2] If we consult Graves,[3] the first to give us a graphic description of reflex paraplegia, we find that he says, on this subject: "With respect to sensation, it appears to be affected as well as motion. In the slow and chronic form of this species of paraplegia, it does not attract the attention of the patient so quickly as the derangement of muscular power; it is generally some time before he notices any diminution of sensation, and then accidentally. In the more advanced stage, however, this becomes manifest, and is accompanied by a feeling of cold in the lower limbs, which seldom extends higher than the knees. In the more rapid and acute form, the derangement of sensation is much more obvious, and is generally the first symptom noticed by the patient. There is at first a feeling of *numbness*, which commences in the toes or feet, and extends up the limb; this, in the course of a few days, is followed by *formication* and *tingling pains* in the course of the nerves, and then loss of power and *diminished sensation*. There is, however, in both these forms of paraplegia (urinary and from cold and wet), much less impairment of sensation than of motion, and the loss of sensation is never so complete as in paraplegia from disease of the spine."

Most of Stanley's cases point out some trouble with the sensibility of the limbs, which once (Case VI.) "was lost, extending up to the navel."[4]

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[1] Op. cit., p. 383.

[2] Op. cit., p. 378.

[3] Op. cit., p. 361.

[4] Medico-Chir. Trans., 1833, Vol. XVIII., p. 260.

R. Le Roy, d'Etiolles, has collected fifty-three cases in his interesting work "On Paralysis of the Lower Extremities." A perusal of these cases, including those of Stanley and Graves, will convince at once that it is the rule to meet, especially in urinary paraplegia, with the very symptoms said not to be present by Brown-Séquard. I translate, concerning the state of sensibility, the conclusions drawn by Le Roy, d'Etiolles. He states, first, with reference to paraplegia from acute myelitis:

"In this, more than in paraplegia from chronic nephritis, there is either complete paralysis of movement and sensibility in the limbs—as with some of the foregoing instances—or, as it frequently happens, movement and sensibility are obviously diminished, the movement more than sensibility, and almost always, in an unequal degree, with both limbs. Thus, the faculty of feeling and of moving may be simply blunt in one limb and totally lost in the other. The weaker limb is sometimes the seat of very violent pain." "Paralysis is frequently preceded by *numbness* in the limbs; *formication* follows it, and continuing along with the nervous derangement; general trembling may be added to these symptoms." On paraplegia from chronic nephritis, he says: "Tactile and painful sensibility has been unimpaired in half of the cases, being *only diminished* with the others, and twice *totally lost*. Once, there was partial *anœsthesia* with *hyperœsthesia* on another portion of the same limb. Tactile and painful sensibility were undamaged on the right limb; on the left there was *anœsthesia* and *analgesia*, principally over the instep; but above the external malleolus there was a very sensitive hyperæsthetic spot, as large as the palm of the hand. In the case of Prof. Sanson, *all sensibility was lost* in both lower limbs, the abdominal walls, and the thoracic, up to near the nipple. In another case, *sensibility was duller* on the right than on the left side, it being necessary to pinch very violently to produce

a shadow of pain."[1] Further on, comparing it with myelitis, he says: "It must be acknowledged that urinary paraplegia does also come preceded by *formication, cramps, convulsive jerkings;* such onset is mainly met with when acute nephritis is the initial cause of the disease."

The several cases collected by Gubler, in the papers referred to, give further evidence that sensibility does not remain unimpaired in cases of reflex paralysis after acute diseases. As regards hysterical paralysis, its peculiar inordinate sensibility is too well acknowledged to need any reference to it. With infantile paralysis, it is difficult to ascertain the true state of sensibility in the paralyzed limbs. Frequently, however, we observe more or less hyperæsthesia, particularly in the early stages of the disease. This hyperæsthetic condition has been noticed, among others, by West,[2] and together with the incomplete loss of power, which allows the child to move instinctively the limb to guard against pain, is one of the causes of overlooking the paralysis in its inception. That peripheral paralysis often gives rise to hyperæsthesia may be ascertained from the several cases reported by Drs. Mitchell, Moorehouse and Keen, in the above mentioned monograph. The case borrowed from Le Bret is also a good example of its occurrence.

If I have insisted on the derangement of sensibility with reflex paralysis, it is on account of the strong evidence it bears upon the part which the sympathetic takes in the production of the paralysis. The history of those cases reported by various authors, and the investigation of others which have fallen under my own observation, convince me not only of the frequency of such trouble with sensibility, peculiar to disturbances of the sympathetic, but also of chills, or cold sen-

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[1] Op. cit., p. 144.

[2] Diseases of Infancy and Childhood, London, 1859, p. 233.

sations, followed by more or less perspiration, especially at the outset of the paralysis. In many instances, the patient has true fever, which, in the majority of cases, is the forerunner of infantile paralysis. The occurrence of chills on the commencement of reflex paralysis is noticed particularly by Graves, Romberg and others.

I could mention the recent curious example of a Mexican, who, upon being kept all one night in a cold and damp prison, had, the next day, facial paralysis on the left side. The attack began with violent, prolonged chills, and pain with numbness extending throughout the arm to the fingers. He continued to have regularly a paroxysm of intermittent fever every morning, with exacerbation of the paralytic symptoms. He had never had ague. It is undeniable that complete anæsthesia rarely exists with reflex paralysis, excepting, however, with hysterical paralysis. Notwithstanding, anæsthesia is associated, oftener than is suspected, though in an incomplete and concealed manner, with reflex paralysis. There are paralytics who cannot stand with closed eyes without tottering or immediately losing balance and falling. Some authors look upon this sign as indicative of lesion in the gray substance of the cord. I have, on repeated inquiry, ascertained that the phenomenon is met with whenever anæsthesia of the lower extremities be present. The greater the anæsthesia, the more marked and sudden will be the tendency to fall. The nature of the paralysis is immaterial for the occurrence of the phenomenon. I have found it so in hysterical paraplegia, in progressive locomotor ataxy, in urinary paraplegia, and, recently, in a case of paralysis of the left leg from injury to the sciatic nerve. In this instance there was atrophy of the thigh and leg, with anæsthesia of the foot, always numb. The patient could stand perfectly well on the right leg with the eyes shut; but no sooner would he attempt to do the same, standing on the left leg, than he would lose

his equilibrium and fall. In an instance of progressive locomotor ataxy, in which the feet had preserved unimpaired sensibility, the patient, a young man, could stand with closed eyes without noticing any change.[1] In two other instances I have observed this curious phenomenon to occur with hyperæsthesia instead of anæsthesia in the feet. Both were cases of chronic spinal meningitis. Whether the sign has the special value asserted by some authors I will not venture to discuss—although anæsthesia and lesion of the spinal gray substance are far from always accompanying each other.

As to hyperæsthesia, I have generally noticed that it has existed with formication and tingling sensations in the limbs. I have also observed that hypodermic injections of strychnia, besides improving the condition of the muscles, rapidly remove the uncomfortable feelings produced by this deranged state of sensibility—more apt to exist with urinary, or uterine and dysenteric paraplegia, infantile paralysis, and with paralysis from wet and cold. In 1864 I had a case of reflex paraplegia at the Central Park U. S. A. General Hospital in this city. The patient, after being all night on picket duty, in water up to his knees, became paraplegic. He complained of a great deal of formication and numbness in the feet; the limbs were cold, bluish, and had lost tactile and painful sensibility up to the knees. The patient could be pricked, pinched, and burned in both legs, without being conscious of it; yet he would complain of constant, deep-seated pain in the feet and legs. This phenomenon, *i. e.* pain along with anæsthesia, I have found in other cases of reflex paralysis, and, not unfrequently, the first causes the latter to be overlooked. Application of electricity, and the internal use of strychnia, slowly relieved the paralyzed condition of the legs, until I injected, hypodermically, one-sixtieth of a grain of strychnine in each limb.

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[1] See note, p. 59.

This operation was repeated four or five times, and, from the first injection, the patient, to his great joy, could walk better, and found himself eased of formication in the legs, which finally recovered their power. The prompt results obtained in this case were witnessed by Dr. G. S. Winston and my other colleagues in the hospital. The following instance shows again the beneficial effects of strychnia, employed in this hypodermic way. A gentleman, after sudden exposure to cold, became paralytic in the right leg. The paralysis, from the onset, was associated with pain, which soon became so intense that the patient lost all rest, being in constant agony. It was thought that he had articular inflammation, and blisters over the hip joint, and narcotics, were freely resorted to, but with slight temporary relief. Meantime the muscles of the thigh and leg began to waste, the hyperæsthesia persisting to such a degree that the slightest touch of the skin of the leg would give him great pain. In this intolerable condition, he determined, on his own accord, to try electricity, and felt some comfort upon its application. He consulted me at this time. The right limb was wasted, cold, and still very sensitive, especially on the anterior and external parts of the leg. I continued the use of electricity, after advising the patient to have one-fiftieth of a grain of strychnine injected into the thigh. He consented to the operation, and the relief it produced was soon manifest. Upon repeating this injection three times more, at intervals of four days, the state of sensibility was quite natural. A tonic regimen, together with electricity and the hot and cold douche to the limb, gradually returned power to the paralyzed muscles. My friend Dr. T. G. Thomas saw this patient and the results of the above treatment from the beginning. Recently, I removed the arthritic pains in dysenteric paraplegia by hypodermic injection of strychnia. I frequently resort to it in infantile paralysis, with the twofold purpose of favoring return of power to the paralyzed muscles, and of removing pain or the

uncomfortable feelings from the limbs, by the local action of strychnia. The useful assistance I have derived from a cautious employment of this means, especially in cases of infantile paralysis, has been witnessed, among others, by my friends Drs. Van Buren, Sabine, Sayre, and Thomas.

I wish to point out a very curious fact, nowhere noticed, as far as I have been able to ascertain, except in two cases of reflex paraplegia, related by H. N. Victor Robert,[1] viz., permanent contraction in the muscles of the paralyzed limbs. This contraction—which may, without paralysis, be one of the sequelæ of typhoid fever—I have met with in a young man, who had paraplegia also, consecutive to typhoid fever, and in a woman with uterine paraplegia, attended with great tenesmus. I, of course, do not refer to the less infrequent contraction of muscles antagonistic to those involved in local paralysis. In the cases cited the contraction was not general with the muscles of the lower limbs; it mainly existed in the feet, keeping the toes strongly flexed. Extension of the contracted muscles was painful; however, by gently exerting it, the toes could be relaxed, to resume afterward their primitive condition. In either instance there was a marked degree of anæsthesia, which, together with the contraction, was cured by local application of galvanic electricity. A slight contraction in some of the paralyzed muscles has been noted by Brown-Séquard, in a boy five years old, who had paraplegia at the very beginning of the second dentition, increasing and decreasing alternately at the time of and after the cutting of each of three molars.[2] Friedberg,[3] under the title of *myopathia dyscrasica*, reports the following interesting case, showing a muscular lesion without derangement of the nerv-

[1] De la Paraplégie consécutive a la Fièvre Typhoïde, Thèse, 1862, pp. 18, 24.

[2] Paralysis of the Lower Extremities, Phil., 1861, p. 16.

[3] Pathologie und Therapie der Muskellähmung, Leipzig, 1862, Obs. IX., and Jaccoud, op. cit., p. 395.

ous system as the only cause of paraplegia following typhoid fever. A girl, sixteen years of age, five weeks and a half after being cured of typhoid fever, was taken with pain in the lower limbs, which disappeared to be replaced by deep-seated numbness, without impairment whatever of cutaneous sensibility, and with convulsive jerkings of the legs, lasting a few days. The patient could not walk nor stand on her feet, nor leave her bed, on account of permanent flexion of the legs on the thighs. There were no signs of muscular atrophy or articular lesion in the lower limbs. Electric sensibility and contractility were lessened in the extensor muscles of the legs. Being in such a condition, the girl was taken, after a meal, mainly of potatoes, with symptoms of intestinal perforation, and in twenty-four hours died. On post mortem examination there were found portions of food in the peritoneal cavity, which had passed through a lacerated cicatrix at the level of the middle third of the jejunum. The brain, spinal cord, and nerves were quite sound, but the extensor muscles on both legs exhibited a remarkable alteration, consisting of suggillations and small abscesses, some containing liquid, others solid pus, while in other places the primitive muscular fibres had undergone a granular fatty degeneration. The coats of the capillary vessels in the triceps of the right side were infiltrated with fat.

I will state, finally, concerning the muscles, that I have found them in infantile paralysis unresponsive to the electric current, without being, however, in a condition of fatty substitution, as admitted, under such circumstances, by Duchenne de Boulogne. I should add, in evidence, that in cases of reflex paralysis the application of an induced current to muscles distant from those of the affected regions, often determines contraction of those which did not respond to the direct application of electricity. I have noticed this phenomenon especially in infantile paralysis, when faradization of muscles of the ante-

rior femoral or of the gluteal region has determined manifest contraction of the gastrocnemius, or of the extensor digitorum, which did not appear on the current being directly applied to these very muscles. As to the muscular structure, a fatty degeneration of the fibres with their interspaces filled by fatty globules has been peculiar to their paralytic condition, while another granular change, reducing the fibres to their myolemma, seems to be present in the contracted muscles. Muscular degeneration in paralysis coexists with more or less impaired nutrition of the ganglionic system and structural changes consequent thereon. Repeated microscopical examinations of my own, as well as those of others, lead me to this belief. As long as the ganglion remains uninjured, and the nutrition continues normally in the peripheral nerve, the muscles escape atrophy. For this reason it is so doubtful that atrophy occurs from purely cerebral disease, as advanced by Drs. Mitchell, Moorehouse and Keen in the work above alluded to. The obstinacy of infantile paralysis, in my opinion, depends principally on the rapid degeneration of the ganglia, which cannot recover their trophic power, and hence degeneration of the nerve and of the muscles and bones. However, I do not pretend to deny by this statement the occurrence of muscular atrophy without original lesion of the nervous system.

The absence of rigor mortis in the palsied limbs, remarked in Cases II. and III., has also been noticed by Charcot. Brown-Séquard[1] says that cadaveric rigidity comes later and lasts longer after section of the sympathetic; but as far as I know the above fact has been pointed out only by Charcot.[2]

The urine, in the cases of reflex paralysis I have examined, has been rather above the average density. It has been found generally acid, and more or less abundant with phosphates and

[1] Physiology and Pathology of the Central Nervous System, 1860, p. 141.

[2] Comptes Rendus de la Société de Biologie, 1863, 3ème Série, Tome V, p. 192.

uric acid. In a good number of cases it has also been neutral, without the existence of retention. I have never detected sugar or albumen in it. Since Dr. Andrew Clarke, of the London Hospital,[1] called attention to the violet color which acid urine may take upon the addition of Trommer's test for sugar, and showed that it was due to the presence of some volatile substance—most probably ammonia—I have had two opportunities of examining such phenomenon in acid urine from subjects with reflex paralysis (a child and an adult). In either case I have met with the results announced by Clarke, on using Trommer's test. I did not, however, detect the presence of bile existing in Clarke's cases, nor in the urine from another patient with neuralgia, likewise acid and with the same reaction.

These researches do not certainly include post mortem examinations of every variety of reflex paralysis. They, however, make reference to cases which have been hitherto considered as typical. Concerning the alterations which I have noted on the sympathetic system, I abstain from venturing any absolute opinion on their bearing, until I have had greater opportunities of comparing these facts with the results of researches which I have already undertaken on the pathological anatomy of the ganglionic system. No doubt that with searching eye the microscope will continue to map out more accurately the morbid alterations of the nervous system in reflex paralysis. Unless a more than heretofore careful registration of a greater number of cases should prove that the cadaveric lesions here described were exceptional, the fact of peripheral and central nervous alterations peculiar to reflex paralysis will remain unquestioned, making the theory of disease without any departure whatever from the normal structure of anatomical elements still more untenable.

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[1] Medical Times and Gazette, June 11, 1864, p. 654.

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